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The Journal of Thoracic Surgery

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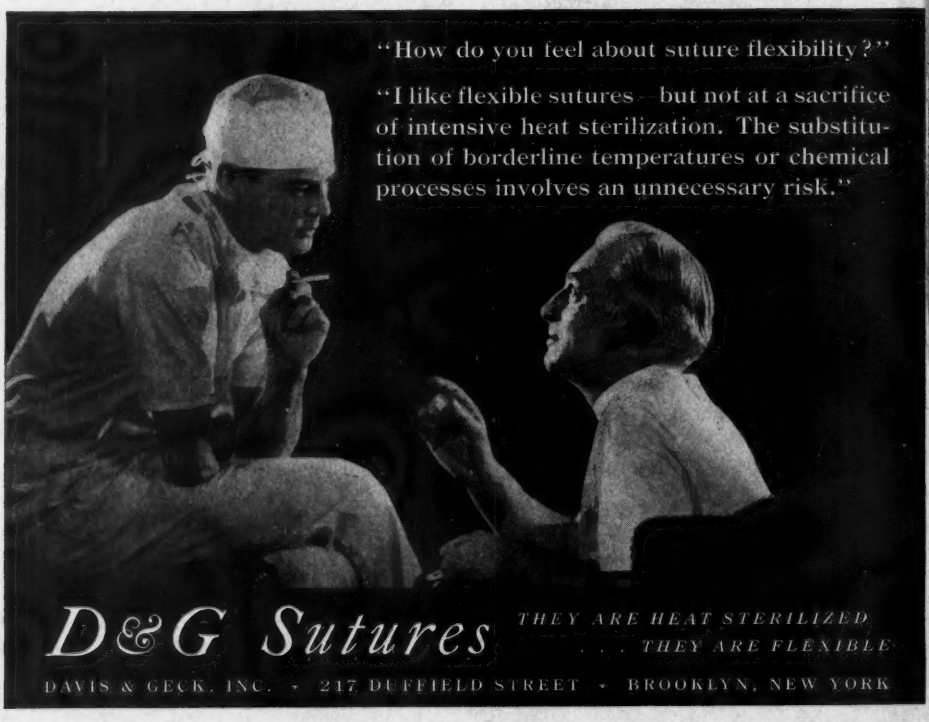
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The Journal of Thoracic Surgery

VOL. 3 FEBRUARY, 1934 NO. 3

Original Communications

THE FUTURE SURGICAL STATUS OF THE COLLAPSE THERAPY PATIENT*

THOS. J. KINSELLA, M.D., F.A.C.S.†
OAK TERRACE, MINNESOTA

THE increasing use of collapse therapy measures in the treatment of pulmonary tuberculosis, bronchiectasis, lung abscess, chronic empyema, etc., adds to our population each year a large number of persons with an impaired breathing mechanism and very frequently a markedly diminished respiratory reserve both from the disease itself and from the surgical procedure. The question of the durability of these persons and of their ability to withstand the strain of ordinary life, with its attendant demands on their respiratory system during both physiologic and pathologic states, is an interesting one.

These patients are subject not only to the ordinary diseases which may attack the average person, but, in addition, the tuberculous group in particular, but to a lesser extent the others also, may be heir to any of the complications or metastatic implantations of their original disease, a number of which may be of such a nature as to demand surgical intervention. The general surgeon, and particularly the surgeon doing thoracic work, should be interested in the reaction of these patients to extrapulmonary surgery and in their ability to withstand various types of surgical procedures. It is with the hope that surgeons in general may feel more confident in undertaking necessary surgery upon patients of this group that the following rather limited experience with extrapulmonary surgical procedures following collapse therapy in a group of tuberculous patients is here presented.

*Read at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

†Assistant Professor of Surgery, University of Minnesota, Surgeon and Assistant Medical Director, Glen Lake Sanatorium.

UNILATERAL PNEUMOTHORAX

The unilateral pneumothorax series includes a group of 22 patients who were submitted to various major extrathoracic surgical procedures, including appendectomy, salpingectomy, uterine suspension, perineorrhaphy, and three types of orthopedic procedures; i.e., an Ollier resection of the tarsus for tuberculosis, a subtrochanteric osteotomy for tuberculosis of the hip, and a spine fusion for tuberculosis of the lumbar vertebrae. This surgery was performed at various times ranging from two months to five years following the institution of artificial pneumothorax. The underlying pathologic process in 9 of these patients was tuberculous in nature and in the remainder, nontuberculous. One of these patients in addition to having tuberculosis and an artificial pneumothorax was a total diabetic. Another patient in addition to the pneumothorax had at the same time, a contralateral temporarily paralyzed diaphragm.

The types of anesthesia used included local infiltration, paravertebral block, nitrous oxide and ethylene in combination with local anesthesia, nitrous oxide and ether, and spinal anesthesia. All of the patients operated upon under spinal anesthesia tolerated their anesthesia very well, with no respiratory embarrassment. Cyanosis occurred more frequently under nitrous oxide anesthesia than with the other types and relaxation was correspondingly imperfect. Ethylene proved more satisfactory in both respects when general anesthesia became necessary.

One patient died six weeks following laparotomy and appendectomy from tuberculous enteritis, salpingitis, peritonitis, and pericarditis. One had a definite spread of pulmonary tuberculosis in the contralateral lung within six months following a laparotomy performed under nitrous oxide and ether. Another operated upon under spinal anesthesia, had a temporary increase in pulmonary symptoms with no x-ray evidence of spread of the disease. Two patients developed pleural effusions within three months following their operations. Two, on the other hand, showed improvement in an unstable lesion in the contralateral lung within the three months following the surgical intervention and in both of these cases the surgery had been directed at a tuberculous focus. Two other patients carrying unilateral pneumothorax were subjected to limited upper thoracoplasty on the contralateral side under local infiltration anesthesia and in both instances this was well tolerated.

Our general sanatorium experience with tuberculosis and pregnancy has taught us to treat the tuberculosis and allow the pregnancy to continue. Two patients are listed in the unilateral pneumothorax group in whom so-called therapeutic interruptions of pregnancy were performed in 1925 and 1930 outside the sanatorium. Twelve patients

TABLE I

MAJOR SURGERY DURING UNILATERAL PNEUMOTHORAX

CASE	INITIAL	AGE AT ONSET OF PNEUMO.	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	F. N.	5 mo.	3/22/25	N_2O Local	–	Appendectomy	Satisfactory
2.	S. S.	54 mo.	3/23/26	Local	Tbc.	Appendectomy	Satisfactory
3.	G. P.	21 mo.	1/ 9/27	Local and N_2O	–	Appendectomy	Satisfactory
4.	C. S.	10 mo.	5/ 6/27	N_2O	–	Appendectomy; suspension uterus	Satisfactory
5.	M. W.	3 mo.	9/15/28	Local and ethylene	Tbc.	Appendectomy	Died 11/1/28. Pul. tbc.; tbc. enteritis; tbc. peritonitis
6.	M. H.	17 mo.	11/ 4/28	Local	–	Appendectomy	Satisfactory
7.	J. B.	42 mo.	4/10/29	Spinal	–	Appendectomy; salpingectomy	Satisfactory
8.	G. L.	25 mo.	6/15/29	Spinal	–	Appendectomy	Satisfactory
9.	M. M.	24 mo.	8/ 1/30	N_2O Ether	–	Hysterotomy; salpingectomy	Spread of tbc.
10.	F. O. M.	29 mo.	12/13/30	Spinal	–	Appendectomy	Increase of symptoms; x-ray no change
11.	V. M.	18 mo.	4/ 8/31	Spinal	Tbc.	Ollier resection of tarsus	Satisfactory
12.	M. C.	7 mo.	8/21/31	Spinal	–	Appendectomy; oophorectomy	Satisfactory—total diabetic
13.	T. M.	2 mo.	12/ 1/31	Spinal	Tbc.	Subtrochanteric osteotomy	Satisfactory
14.	O. P.	21 mo.	3/31/32	Spinal	–	Appendectomy	Satisfactory
15.	L. F.	33 mo.	8/25/32	Spinal	–	Drainage appendiceal abscess	Satisfactory
16.	M. S.	60 mo.	11/29/32	Spinal	Tbc.	Salpingectomy; appendectomy	Improved chest; contralateral phrenic
17.	M. A.	33 mo.	2/ 2/33	Spinal	Tbc.	Appendectomy	Satisfactory
18.	M. F.	11 mo.	2/ 8/33	Spinal	–	Suspension of uterus; perineorrhaphy	Satisfactory
19.	A. W.	12 mo.	2/18/33	Spinal	Tbc.	Appendectomy	Satisfactory
20.	O. R.	13 mo.	2/28/33	Spinal	Tbc.	Appendectomy	Bowel hemorrhage ceased
21.	I. J.	18 mo.	3/26/33	Spinal	Tbc.	Appendectomy	Satisfactory
22.	V. M.	42 mo.	4/25/33	Ethylene	Tbc.	Fusion tbc. spine	Satisfactory
23.	S. S.	96 mo.	8/29/29	Local	Tbc.	Thoracoplasty opposite	Well tolerated
24.	M. T.	11 mo.	3/21/29	Local	Tbc.	Thoracoplasty opposite	Well tolerated

TABLE II

PREGNANCY DURING UNILATERAL PNEUMOTHORAX

CASE	INITIAL	AGE AT ONSET OF PNEUMO.	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	A. B. L.	22 mo.	5/28/27	N_2O	–	Delivery	Clear fluid changed to tbc. empyema; cleared; resolution opposite
2.	M. M.	2 mo.	10/29/28	O	–	Delivery	Fluid 6 mo. later
3.	P. S.	29 mo.	2/ ?/29	C H Cl_3	–	Delivery	Satisfactory 17 mo.; then second preg. and spread of tbc.
4.	F. M.	96 mo.	11/11/29	?	–	Delivery	
5.	C. W.	35 mo.	2/16/30	?	–	Delivery	Satisfactory
6.	N. S.	4 mo.	8/26/30	C H Cl_3	–	Delivery	Pleural effusion developed
7.	F. W.	2 wk.	3/24/31	N_2O	–	Delivery	Satisfactory
8.	K. H.	2 mo.	7/ 3/31	Spinal	Tbc.	Cesarean section; appendectomy	Died 9/4/31; tbc. prog.
9.	A. H.	5 mo.	8/25/32	N_2O	–	Delivery	Satisfactory
10.	D. W.	84 mo.	11/10/32	?	–	Delivery	Satisfactory
11.	S. K.	12 mo.	3/15/33	N_2O? Ethylene	–	Delivery	Satisfactory; phrenic same side
12.	V. M.	32 mo.	2/ 3/33	N_2O	–	Delivery	Satisfactory

MISCELLANEOUS MINOR PROCEDURES DURING UNILATERAL PNEUMOTHORAX

22		1 to 72 mo.		Local	Tbc. 7 – 15	Tonsillectomy	
21		1 to 13 mo.		Spinal 12 Local 9	Tbc. 15 – 6	Rectal operations	
9		2 to 35 mo.		Spinal 4 Local 5	– 9	Gynecologic and genito-urinary operations	
5							
—		5 to 17 mo.		Local 5	– 5	Miscellaneous minor operations	
93							

in this same group went through pregnancy and delivery at term. Of these 12 pregnancy patients, 7 had been taking pneumothorax for periods of time varying from one to seven years before delivery. Five of them had had the artificial pneumothorax established during pregnancy, from two weeks to five months before delivery. One patient had had a permanent phrenic nerve operation performed before the institution of the pneumothorax. Another, who had been taking pneumothorax for two months, was delivered under spinal anesthesia by cesarean section and at the same time had a tuberculous appendix removed. This woman, who had a far-advanced bilateral progressive pulmonary tuberculosis at the time of admission, died two months later from tuberculosis. All of the others were delivered spontaneously at term and the majority, even the primipara, had rather easy labors. All are still living.

One of these patients, who was carrying a clear pleural effusion during her pregnancy and was showing a slight increase in the tuberculosis in the contralateral lung, developed a tuberculous empyema following delivery and immediately began to clear the contralateral tuberculosis. The tuberculous empyema eventually cleared up spontaneously. She is still taking pneumothorax and is in good condition. Another patient whose pneumothorax had been started four months before delivery, formed a clear pleural effusion one month following labor and three months later developed a tuberculous empyema. Subsequently an extrapleural thoracoplasty obliterated this pyopneumothorax. A third, whose pneumothorax had been started in the seventh month of pregnancy, developed a clear effusion six months following delivery. This subsequently cleared and she is still continuing her pneumothorax. A fourth patient remained in good condition for seventeen months following delivery and then, during a second pregnancy, developed a spread of her tuberculosis which necessitated a bilateral pneumothorax. She appears subsequently in the bilateral pneumothorax series. Three of these women were delivered without anesthesia, five under nitrous oxide, one under nitrous oxide combined with a small amount of ether, two under light chloroform narcosis, and one by cesarean section under spinal anesthesia. The anesthesia was well tolerated in all cases.

Twenty-two patients, in addition to the above mentioned groups, were submitted to tonsillectomy under local anesthesia from one month to six years following the institution of their pneumothorax, 7 for tuberculous and 15 for nontuberculous tonsils. Twenty-one patients were subjected to rectal operations of various types for ischiorectal abscess, fistulas, etc., at times varying from one to thirteen months following the institution of their pneumothorax. Fifteen of these were tuberculous and 6 had nontuberculous lesions. Nine operations were done under local infiltration and 12 under low spinal anesthesia. Nine

others had minor gynecologic or genitourinary operations for nontuberculous conditions from two to thirty-five months following the institution of pneumothorax. Four of these were done under low spinal and 5 under local infiltration anesthesia. Five patients had various minor operations under local infiltration anesthesia for nontuberculous lesions, from five to seventeen months after the institution of their pneumothorax. One patient, six weeks following a tonsillectomy under local anesthesia for tuberculous tonsils, developed an increase in the tuberculous lesion in the lung.

BILATERAL PNEUMOTHORAX

The bilateral pneumothorax series is, of course, considerably smaller, including only 5 patients. The first had an appendectomy for a nontuberculous appendix performed under local infiltration anesthesia six months after the institution of the second pneumothorax.* The second patient was delivered by cesarean section under spinal anesthesia four and one-half months after the institution of the second pneumothorax and one week following a pulmonary hemorrhage. Four days later a second spinal anesthesia was given for the removal of retained membranes. The third patient was operated upon for a tuberculous ischiorectal abscess under low spinal anesthesia four and one-half months after the institution of the second pneumothorax. The other two had tonsillectomies under local anesthesia, one for tuberculous tonsils, one two months and the other thirty-eight months following the establishment of the second pneumothorax.

TABLE III

SURGERY DURING BILATERAL PNEUMOTHORAX

CASE	INITIAL	AGE AT TIME OF SECOND PNEUMO.	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	A. I.	6 mo.	1/19/28	Local	–	Appendectomy	Satisfactory
2.	P. S.	4½ mo.	2/27/31	Spinal	–	Cesarean section;	Satisfactory
			3/ 1/31	Spinal		removal of membranes	
3.	R. B.		2/26/32	Spinal	Tbc.	Rectal surgery	Satisfactory
4.	E. T.	38 mo.	5/18/28	Local	–	Tonsillectomy	Satisfactory
5.	L. R.	2 mo.	11/ 4/30	Local	Tbc.	Tonsillectomy	Satisfactory

The spinal anesthesia for the cesarean section was extended to the costal margin on both occasions, while for the rectal surgery a low spinal with anesthesia to the umbilicus only was used. The anesthesia was perfectly satisfactory, was well tolerated by the patient, and was not accompanied by dyspnea or cyanosis. No difficulty was encountered either during or following the operations and no complications occurred.

*Since this paper was presented, two additional patients with bilateral pneumothorax were subjected to appendectomy for acute appendicitis, under spinal anesthesia, without complications and with satisfactory results.

PHRENIC NERVE SURGERY

This group consists of 8 patients upon whom nine major extrapulmonary operations were performed at times varying from one to forty-two months following the phrenic nerve surgery. Six additional patients who were subjected to major surgery following phrenic nerve surgery will be included in the thoracoplasty series. The surgical procedures included: appendectomy, dilatation and curettage, salpingectomy, an operation for the relief of intestinal obstruction, a short-circuiting operation upon the bowel, ureterolithotomy, palliative resection of a carcinoma of the stomach and an exploratory laparotomy for an intestinal obstruction due to a retrograde intussusception of the jejunum into the stomach through a gastroenterostomy stoma. Three of these operations were performed for tuberculous and 6 for nontuberculous lesions. All operations were performed under spinal anesthesia, with two supplemented by nitrous oxide and ethylene. One patient of this group was carrying a pneumothorax partially collapsing the contralateral lung.

Five other patients were subjected to rectal surgery under low spinal anesthesia. Fourteen others had nose and throat operations, rectal surgery, gynecologic surgery, and various other minor procedures under local anesthesia. The pathologic process was tuberculous in 6 and nontuberculous in the remaining 13 cases. Two patients carrying paralyzed diaphragms went through pregnancy and delivery without difficulty, one at twenty-six and the other at thirty-two months following the phrenic nerve operation. The second of these patients had a supplementary pneumothorax on the same side.

Three patients listed in this series died following their operations. The first died twenty-four hours after an exploratory laparotomy for an intestinal obstruction due to a retrograde intussusception. The lesion, fatal in itself, was not amenable to surgery at the time explored. The second patient died seventy-two hours after a ureterolithotomy performed under spinal and nitrous oxide anesthesia, from a fulminating infection in the wound, with no evidence of pulmonary complications. The third patient died fourteen days following a palliative resection of a bleeding carcinoma of the stomach, the death in this case being due, according to the pathologist, to an activation of tuberculosis in the lung on the same side as the phrenic operation. The peritoneal cavity was negative. It does not seem, from postmortem examination or clinical study, that the paralysis of the diaphragm was in any way a contributing factor to the death of these patients.

One patient in this series, following an appendectomy, experienced a great deal of trouble expelling sputum from the chest, a complication which I believe is the result of previous phrenic nerve surgery on this side. The same difficulty has been noticed on a number of occasions in

TABLE IV

SURGERY AFTER PHRENIC

CASE	INITIAL	TIME SINCE PHRENIC	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	N. C.	8 mo.	6/13/29	Spinal	Tbc.	Intestinal obstruction; intestinal anastomosis	Satisfactory
		14 mo.	1/17/30	Spinal	Tbc.		Satisfactory
2.	K. H.	2 mo.	9/25/30	Spinal	–	Intestinal obstruction; retrograde intussusception	Died 24 hours
3.	A. B.	6 mo.	9/ 7/32	Spinal and N_2O	–	Ureterolithotomy	Died 72 hours; infected wound
4.	L. G.	1 mo.	10/ 7/32	Spinal and ethylene	–	Palliative resection; carcinoma of stomach	Died 14 days; tbc. pneumonia
5.	M. S.	17 mo.	11/29/32	Spinal	Tbc.	Double salpingectomy; appendectomy	Pneumo. opposite chest—improved
6.	B. S.	42 mo.	12/ 2/32	Spinal	–	Dilatation and curettage	Satisfactory
7.	L. B.	41 mo.	4/ 7/33	Spinal	–	Appendectomy	Satisfactory
8.	E. R.	37 mo.	4/16/33	Spinal	–	Appendectomy	Satisfactory
				PREGNANCY AFTER PHRENIC			
1.	E. C.	26 mo.	11/ 4/30		–	Delivery	Satisfactory
2.	S. K.	32 mo.	1/ ?/33		–	Delivery	Satisfactory—pneumo. same side
				MISCELLANEOUS MINOR PROCEDURES AFTER PHRENIC			
5		6 to 13 mo.			Tbc. 1 – 4	Nose and throat operations	
6		3 to 12 mo.		Spinal 5 Local 1	Tbc. 5 – 1	Rectal surgery	
4		3 to 22 mo.			–	Gynecologic surgery minor	
4		1 to 23 mo.			–	Miscellaneous minor surgery	
29							

thoracoplasty work on patients who had previously had a paralyzed diaphragm and who still had considerable daily sputum. Clinically, it would seem that some of these patients had more difficulty in expelling secretions from the lung than do patients who had not had preliminary phrenic nerve surgery. Aside from the points mentioned, no difficulty has been encountered in this series. All withstood anesthesia to the costal margin or slightly above without embarrassment.

SURGERY FOLLOWING THORACOPLASTY

Fifteen patients with extrapleural thoracoplasty were subjected to twenty operative procedures of major importance at times varying from two and one-half to forty-one months following the completion of their thoracoplasty series. The operations included appendectomy, nephrectomy, ureterectomy, hysterectomy, salpingectomy, cholecystectomy, gastroenterostomy, resection of the clavicle, fusion of the wrist, fusion of the ankle, and mastoidectomy. Eight of these operations were performed for tuberculous and 12 for nontuberculous lesions. Spinal anesthesia was used for 13, ethylene for 1, nitrous oxide for 3, nitrous oxide and local for 2, and nitrous oxide and spinal for 1. Six of these patients had had paralysis of the diaphragm either before or after their thoracoplasty series but before this surgery was undertaken. Anesthesia was well tolerated by all in this group. One patient died four days after an operation for a ruptured appendix with a diffuse peritonitis. This patient had been greatly weakened by a previous pneumonia. The clinical cause of death as proved at postmortem, was general peritonitis and bronchopneumonia.

Three of the thoracoplasty patients went through pregnancy and delivery, one recently for the second time, at periods varying from forty-eight to ninety-two months following thoracoplasty. Three of the deliveries were under nitrous oxide anesthesia, the other a spontaneous delivery without anesthesia. A temporary flare-up of the tuberculosis, which subsequently quieted down, occurred in one; otherwise no complications have been encountered.

Five patients have undergone accidental trauma of considerable severity, 4 of them in automobile accidents and 1 by being kicked in the chest by a horse. Four of these patients had one or more ribs on the operated side fractured and one, in addition, sustained a fracture of the clavicle, which healed in malposition necessitating subsequent partial resection. This last patient had a subsequent exacerbation of pulmonary symptoms which required further chest operation before they were controlled. One patient in this group, as the result of falls, had a Pott's fracture of the ankle and a Colles fracture of the wrist. All, at the present time, are in good condition.

The thoracoplasty series includes four additional patients who have had spinal anesthesia for various minor procedures, making a total of

TABLE V

SURGERY AFTER THORACOPLASTY

CASE	INITIAL	TIME SINCE THORACO.	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	M. B.	18 mo.	2/13/27	N_2O	–	Appendectomy	Satisfactory
2.	M. P.	22 mo.	12/12/28	N_2O	Tbc.	Ureterectomy; nephrectomy	Chest satisfactory; phrenic; wound infected—healed
		23 mo.	1/ 5/29	N_2O and local	Tbc.		
3.	M. S.	6 mo.	4/26/28	Spinal	Tbc.	Hysterectomy and salpingectomy	Satisfactory; fecal fistula—healed
		41 mo.	3/25/31	Spinal and local	Tbc.	Fusion wrist	
4.	W. T.	32 mo.	11/ 6/29	N_2O	Tbc.	Fusion wrist	Satisfactory
5.	E. P.	27 mo.	2/ 9/31	Spinal and N_2O	–	General peritonitis; appendectomy	Died 2/13/31; pneumonia; general peritonitis
6.	G. B.	17 mo.	3/11/31	Spinal	Tbc.	Nephrectomy	Satisfactory; phrenic
7.	R. S.	4½ mo.	3/11/31	Spinal	Tbc.	Appendectomy	Satisfactory
8.	M. R.	24 mo.	5/27/31	Spinal	–	Exploratory on kidney	Satisfactory; phrenic
		28 mo.	9/10/31	Spinal	–	Appendectomy	
9.	F. B.	2½ mo.	6/ 4/31	Spinal; local	–	Cholecystectomy	Satisfactory
10.	E. T.	26 mo.	11/12/31	Spinal	–	Appendectomy	Satisfactory; phrenic
11.	A. H.	17 mo.	12/ 7/31	Spinal	–	Fusion ankle	Satisfactory; phrenic
		21 mo.	3/23/32		–		
		30 mo.	1/15/33		–		
12.	L. G.	26 mo.	8/ 8/32	Spinal	–	Gastroenterostomy	Satisfactory
13.	B. D. G.	3 mo.	10/ 5/32	Local; gas	–	Resection clavicle	Satisfactory
14.	R. B.	28 mo.	11/10/32	Spinal	–	Appendectomy	Satisfactory; phrenic
15.	D. S.	13 mo.	1/20/33	Ethylene	Tbc.	Mastoidectomy	Satisfactory

TABLE VI

PREGNANCY AFTER THORACOPLASTY

CASE	INITIAL	TIME SINCE THORAC.	DATE	ANESTHESIA	TBC. OR NON-TBC.	OPERATION	RESULT
1.	B. W.	66 mo.	3/ 3/29	–	–	Delivery	Temporary reactivation of tuberculosis
2.	M. H.	65 mo.	1/13/30	N_2O	–	Delivery	Satisfactory
		92 mo.	5/ 2/32	N_2O	–	Delivery	Satisfactory
3.	E. A.	48 mo.	8/ ?/31	N_2O	–	Delivery	Satisfactory
4.	M. C.	26 mo.	7/ ?/31	Spinal	–	Therapeutic interruption 3 months	Satisfactory; phrenic
				ACCIDENTS AFTER THORACOPLASTY			
1.	B. D. G.				–	Fracture claviele; chest injury; *(auto)*	Recurrence of chest symptoms—reoperated
2.	G. L.				–	2 ribs fractured on operated side *(auto)*	Satisfactory
3.	N. R.				–	1 rib fractured on operated side *(auto)*	Satisfactory
4.	M. B.			Local	–	1 Potts fracture (fall); Colles fracture (fall); rolled by auto *(auto)*	Satisfactory
5.	M. C.				–	2 ribs fractured on operated side; *(kicked by horse)*	Satisfactory
				MISCELLANEOUS MINOR PROCEDURES AFTER THORACOPLASTY			
18				Spinal		Miscellaneous; tonsillectomy	Satisfactory
7		4 to 46 mo.		Local	– 5 Tbc. 2		Satisfactory
2		10 to 40 mo.		Local	–	Nasal	Satisfactory
6		2 to 8 mo.		Caudal and local	– 4 Tbc. 2	Rectal	Satisfactory
2		8 to 36 mo.		Caudal	–	Gynecologic	Satisfactory
6		5 to 47 mo.		Local	– 6	Miscellaneous	Satisfactory
—				Spinal			
47							

19 who have undergone operations under spinal anesthesia. Twenty-three other patients have had miscellaneous minor procedures done under local or caudal anesthesia, 4 of them for tuberculous and 19 for nontuberculous disease. The operations included nose and throat operations, rectal and gynecologic surgery.

The thoracoplasty patients proved themselves to be stable persons who withstood their anesthesia without difficulty and rallied promptly following operation. Under conservative handling they sustained no apparent damage to their tuberculosis.

DISCUSSION

From the small series of cases here studied, it is quite evident that necessary surgical procedures may be undertaken on collapse therapy patients without undue risk. Emergency surgical procedures may be undertaken without delay in such cases the same as in the other tuberculous patients without collapse therapy. Operations of election, however, should be postponed until the patient is well accommodated to the collapse itself and until such a time as the tuberculosis is quiescent, if possible, if the risk is to be held to a minimum.

The question of anesthesia is important. Unquestionably local infiltration anesthesia is the safest method available if the surgeon is schooled in its use and if the contemplated operative procedure lends itself to this technic. Spinal anesthesia has been of great help in the handling of these patients. Probably no group of patients has profited more by its use than those with tuberculosis and, in particular, those with collapse therapy faced by the necessity for surgery. Anesthesia to the fifth or seventh dorsal level was well tolerated by all of this group without any untoward reaction. It should, I believe, be the method of choice for those conditions which can be handled satisfactorily under this type of anesthesia. General anesthesia has been used successfully, but, I believe, should not be used if either or both of the others will suffice. In the presence of active tuberculosis of the lungs, general anesthesia is attended by greater risk of reactivation of the tuberculosis than is the use of either local or spinal, the deep breathing with general anesthesia adding a factor of trauma to the diseased areas in the lung not present with the other types. During quiescent or arrested tuberculosis, this is of less importance, though still a matter to be taken into consideration. Where only light anesthesia is necessary, nitrous oxide will suffice. It is less satisfactory for deeper anesthesia, the diminished respiratory reserve rendering cyanosis more frequent and relaxation less perfect. If deeper anesthesia is necessary or desirable, ethylene is to be preferred, while still deeper anesthesia may be obtained by the addition of a small amount of ether. The use of combined anesthesia, local supplemented by light nitrous oxide or ethylene, is to be preferred to deep general anesthesia, particularly in

the patient with secretions in the chest. Straight ether anesthesia is less desirable and should rarely, if ever, be necessary. Deep anesthesia of any general type to the stage where the cough reflex is obliterated or markedly diminished, is to be avoided, particularly if there are secretions in the chest, if aspiration and spread of tuberculosis is to be prevented. It must not be forgotten, however, that high spinal anesthesia, while not obliterating the cough reflex, does paralyze the muscles used in expelling secretions from the chest and that, consequently, aspiration may occur. It is as essential here as in thoracic work that the lung be emptied of sputum before operation, if possible.

In general, minor surgical procedures have been well tolerated, though in a few instances the patient has been somewhat upset by them and on two occasions, one following tonsillectomy and one following a dental extraction with infection, there was a definite exacerbation of pulmonary tuberculosis.

Major surgical procedures have also been well tolerated by the majority of patients in these four groups and practically as many patients have shown improvement in their chest lesions following their surgery as have developed an exacerbation of their trouble. The 6 deaths which have occurred in this series were more closely related to the original pathologic process present and to the tuberculosis than to the surgical procedure. In at least 4 of them, I believe we can truthfully say that the surgery in no way hastened the end and in none of them do I believe the collapse therapy was of major importance in the fatal outcome. Difficulty in expectoration following operation was encountered in the phrenic nerve series and might be a predisposing factor to pulmonary complications, but thus far this has not been evident in this group. Postoperative hyperventilation of the lungs was not practiced routinely, because we wished to avoid the trauma of deep breathing on the tuberculous lung. Frequent changes of position and active and passive motion of extremities was insisted upon, and all patients were required to raise their daily quota of sputum regularly following all surgery. Exacerbation of pulmonary tuberculosis appeared no more frequently following surgery for tuberculous lesions than for nontuberculous pathologic conditions.

The same principles of surgical handling which should be applied to the average person suffering from tuberculosis should be applied to a greater degree to this group; namely, that we must be more conservative with these patients, we must not whip them too much or lower their resistance too greatly, we must hold trauma to a minimum and select the most favorable time and conditions for the surgery. Tuberculous patients must be given plenty of time for recuperation afterward, so that any tuberculosis which might be reactivated may have a chance to quiet down before the patient is rehabilitated. Anesthesia

should be chosen to suit the patient and the patient's tuberculosis rather than the surgeon. Trauma to active tuberculous disease must be held to a minimum. If surgery can be postponed until the patient's tuberculosis is more or less quiescent or arrested, the risk may be greatly reduced.

CONCLUSIONS

Necessary surgical intervention may be undertaken in collapse therapy patients, the same as in other persons suffering from tuberculosis, if due attention is paid to the cardinal principles underlying the surgical handling of the tuberculous patient and due allowance made for their diminished vital capacity.

ESOPHAGECTOMY FOR CARCINOMA OF THE UPPER ESOPHAGUS WITH LESSONS DERIVED FROM AN OPERATIVE FAILURE*

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IN 1902 Jacobson and Steward[1] stated that esophagectomy for cancer was introduced only to be abandoned, because of the high mortality associated with the operation. The literature on the subject deals largely with carcinoma at the lower end of this structure. Contributions on surgical treatment of carcinoma in the upper esophagus are comparatively meager. According to most authors, the routine treatment should be palliative. Radical surgery is indicated only in a few early cases.[2]

Billroth,[3] in 1870, first demonstrated upon dogs the possibility of esophagectomy for the removal of esophageal neoplasms. In 1877, Czerny[4] performed esophagectomy successfully for cancer in an old woman, but the growth recurred in a year. The technic of esophagectomy was described by Nasiloff[5] in 1880, who used the mediastinal approach on cadavers. Potarca[6] advocated operation upon the right side because of the danger of dealing with the aorta on the left, though the way in which the right pleura naturally dips behind the esophagus makes damage of this structure more likely when the operation is done on the right side. Rehn,[7] who in 1898 attempted to remove a carcinoma of the esophagus, made his approach through the right thoracic wall. Bryant,[8] however, felt that the esophagus should be approached only on the left side above the aortic arch; below this point it should be exposed on the right, and below the ninth dorsal vertebra it is not readily accessible to surgical interference. In 1905 Sauerbruch,[9] after experimenting upon dogs, reported 5 operations for carcinoma of the esophagus, all of which ended fatally; in 2 cases empyema resulted.

Sauerbruch and von Mikulicz[10] summarize the dangers and difficulties attending esophagectomy:

1. It is extremely difficult to resect the esophagus, especially the upper portion. The two ends are not readily reunited after resection. The tube does not stretch and when shortened the consequent tension causes the divided ends to retract. Sutures do not hold. Von Mikulicz never attempted to reunite the esophagus after high resections but closed the gastric end and dropped it back into the wound. The patient was afterward fed through a gastrostomy tube. Twelve cases at the Breslau Clinic up to 1908 all ended fatally.

*Read at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

2. Hemorrhage may be severe during approach through the thoracic wall. All bleeding points must be seized promptly and care taken not to damage the thoracic duct or the vagi.

3. In transmediastinal operations there is great risk of injury to the pleura. While movements of the left lung are impaired, the entry of air into the right pleura may cause death. Sauerbruch always worked in a negative air-pressure chamber.

4. Empyema and infection of the mediastinal cellular tissues are constant hazards. The pleura has little of the tendency of the peritoneum to limit infection, so that even with drainage, infection may be carried in from the outside and an empyema result.

The first successful case of resection of the thoracic portion of the esophagus for carcinoma was done by Torek[11] at the German Hospital, New York, on March 14, 1913. The patient was sixty-seven years old. She died at the age of eighty after a four days' illness of pneumonia, after a postoperative life of thirteen years in comfort and good health. The lesion was below the lower border of the aortic arch. After a preliminary gastrostomy Torek operated transpleurally through an incision in the seventh intercostal space, then extending upward posteriorly, with section of the seventh, sixth, fifth, and fourth ribs near the tubercles. The esophagus was mobilized up to the neck and divided between ligatures and below the carcinoma. The lower stump was invaginated. The upper portion with the mobilized tumor was then drawn out through an incision in the root of the neck and the tumor excised. The thorax was closed without drainage and the redundant portion of the upper esophageal segment placed in a subcutaneous tunnel so that it emerged through a transverse incision in the skin at the level of the second intercostal space. The patient swallowed through a rubber esophagus.

Eggers[12] repeated Torek's surgical feat in 1924. His patient was a man thirty-eight years old who had a squamous-celled epithelioma almost completely closing the esophageal lumen and situated 15 inches from the front teeth. His patient lived in good health about a year. At autopsy a recurrence of the carcinoma was found in the upper part of the stomach involving the pancreas. It was thought by Torek that the recurrence began in the stump and that Eggers had not removed enough healthy tissue below the tumor.

Lilienthal[13] tried Torek's operation six times but was never successful. He secured a complete operative recovery with the retropleural method. The carcinoma, 1½ inches below the aortic arch, was reached by posterior mediastinotomy, mobilization of the esophagus, and the first stage of a skin plastic done with general anesthesia. Two weeks later the esophagus was resected without any anesthesia. Ten months later the tumor recurred in the form of nodules at the junction of the

esophagus and the skin tube. Radium treatment was ineffectual. Death occurred sixteen months after the operation.

Both Torek and Lilienthal ascribed the recurrence to the fact that resection did not extend sufficiently far above the tumor.

These observations emphasize the necessity of operating at a generous distance from the tumor above and below.

Another element essential to success is the avoidance of postoperative infection. One is justified in going to extremes in order to overcome this hazard. The extent to which a surgeon may go to achieve this end is illustrated by Hedblom's[14] successful case reported in 1922. In three stages the fifth to the eleventh ribs were resected on the left side, and three weeks later the esophagus was resected. The graduated thoracoplasties permitted the patient to become accustomed to changed conditions of respiration. During the resection the exteriorization of the location of the proposed stomas in both esophagus and stomach was an important factor in the prophylaxis of infection.

The following case is reported in order to stress the magnitude and gravity of esophagectomy, as well as the liability of postoperative complications.

CASE REPORT

J. W., a fireman, fifty years of age, married, came to our hospital January 19, 1933, complaining of difficult deglutition lasting over a period of three months. On November 11, 1932, he noticed that his food, unless thoroughly masticated, could not be swallowed. His embarrassment gradually increased until he was unable to swallow anything but liquids. There was no pain, but merely the sensation of obstruction in the upper thorax. His previous health was excellent.

Physical examination was essentially negative. The urine and blood were negative. Nonprotein nitrogen was normal, the blood Wassermann negative.

X-ray examination of the esophagus revealed a constriction just below the level of the aperture of the thorax.

Four days after admission a gastrostomy was done. The patient was given a high caloric diet and fluids in quantity through the gastrostomy tube. A rapid improvement in his general condition was observed.

February 4 the esophagus was resected in the following manner:

Approaching the esophagus through the right side posteriorly, a paravertebral incision was made from the second rib to the seventh, then curving forward along the seventh to the midaxillary line. Each rib, from the third to the sixth inclusive, was severed between the angle and tubercle. Digital separation of the pleura from the anterior wall was difficult because this serous membrane is extremely thin and delicate. The pleura was then separated posteriorly and detached from the vertebral column until the neoplasm was entirely uncovered over an area which exposed a wide margin of normal esophagus above and below the growth. The spindle-shaped tumor involved about 7 or 8 cm. of the esophagus just below the aperture of the thorax. (Fig. 1.)

Our first accident occurred at this time. In compressing the lung with a large retractor, an exposed end of a severed rib punctured the pleura, producing a pneumothorax. By pressure anesthesia the lung was not allowed to collapse, yet its expansion was continued to a degree which supplied adequate oxygenation and at the same time made the field of operation more accessible.

The esophagus was separated from its bed and drawn toward the right side with strips of tape. The azygos vein, a surprisingly large vessel, directly in the path of dissection, was tied off and severed. The esophagus was ligated with chromic catgut at two points about 3 cm. below the tumor and severed. The stump on the gastric side was invaginated. Traction on the upper ligature caused it to slip off. There was no discharge from the lumen but some contamination must have occurred before another ligature was applied.

The upper portion of the esophagus including the tumor was mobilized without difficulty and passed upward behind the clavicle and brought out through an incision above the sternoclavicular articulation. There was just enough esophagus to exteriorize the entire tumor and suture the healthy tissue to the fascia. The chest wound was then closed in layers and the right lung completely expanded.

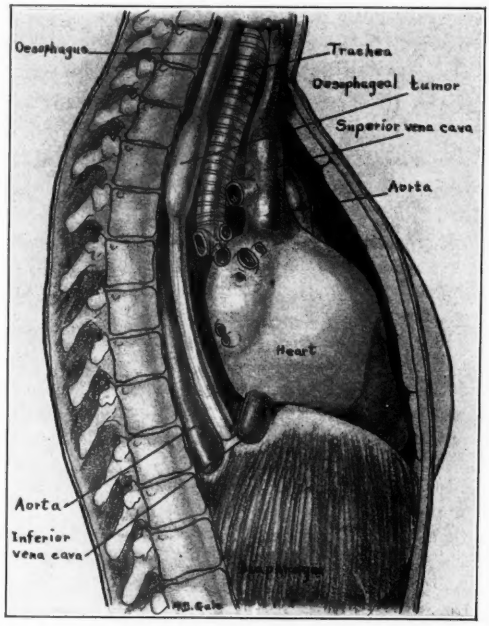

Fig. 1.

Microscopic examination of the tumor revealed it to be a squamous-celled carcinoma.

The patient seemed to do well for four days. Then the temperature rose and continued an irregular elevated range.

On the sixth day an x-ray film showed a fluid level in the right chest. Chest tap yielded a foul brownish fluid. A rib resection was done under local anesthesia, and about 500 c.c. of foul fluid was evacuated. Following this the temperature approached normal. The right lung remained almost completely collapsed. The patient was fed successfully through a gastrostomy tube but resumed a septic type of temperature.

The pleural cavity seemed adequately drained, but it was not possible to reexpand the right lung. The patient was transfused on three occasions, but gradually became weaker and died on the seventeenth postoperative day.

Postmortem examination showed the right lung collapsed and the entire right pleural cavity infected. There were no undrained pockets of pus and the mediastinum was also adequately drained. A subphrenic abscess was found on the right side and, in addition, there was a generalized peritonitis.

REMARKS

There were two accidents in the course of this operation which had special significance. The first occurred during the approach to the esophagus. While retracting the chest wall, the end of one of the severed ribs punctured the pleura. As already stated, this resulted in immediate collapse of the right lung.

The second accident occurred in dividing the esophagus. The proximal ligature did not slip off but actually cut through the structure of the esophagus. Here it is pertinent to point out that the esophagus has four coats: a muscular, a submucosal, a tunica mucosa, and a tunica adventitia, all of soft friable tissue with no connective tissue layers. Breaking through the esophagus with the ligature resulted in the escape of some of the contaminating elements of the tumor and no doubt this accounted for infection in the mediastinum and pleura. To obviate such complications, we feel that two suggestions are worthy of consideration:

1. That the operation be performed in two stages, the first a thoracoplasty consisting of removal of large sections of the ribs from the third to the sixth inclusive. This in itself is a major operation. The approach to the esophagus would be much easier and safer if undertaken at the end of an interval of a week or ten days.

2. That the esophagus be carefully handled near the point of division. To avoid tearing this structure, we would suggest that the proximal side of the line of division be tied with tape and handled with the utmost caution to prevent it from slipping off.

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DISCUSSION

DR. FRANZ TOREK (NEW YORK).—The possibilities of failure are very numerous and in some of my publications I have mentioned a number of such possibilities. Operative accidents may occur on the one hand, and on the other hand the patients' run-down condition may make it impossible for them to stand an operation of that nature. The outstanding obstacles to recovery are the condition of the patient's heart, lungs, and kidneys. Many of these patients are seen suffering from emphysema, weak heart muscle, and any number of other complications which make it impossible for them to go through so serious an operation.

In Dr. Truesdale's case the accident consisted in the slipping of the ligature on the upper stump of the esophagus. In the ligature of the upper stump I have always used a thicker thread than in the ligature of the lower one which is immediately invaginated and so is only of temporary use. For the upper stump a thicker ligature is safer, and Dr. Truesdale, himself, conscious of that fact, recommends the use of tape instead of ligature. The idea is good, and I would recommend its use, especially if the condition of the esophagus is such that it might be cut through by a silk thread.

The access, which in this case was through the sixth intercostal space with division of some ribs higher up, is, in my opinion, the best approach. The only other possibility is to use Sauerbruch's operation, that of an approach from the front by an incision along the clavicle, then down on the sternum and thence outward again, raising the corresponding part of the chest wall. Sauerbruch has had an operative success with the anterior exposure and so, of course, that must be considered as one of the recognized ways of approach. I want to congratulate Dr. Truesdale on his exposition of the difficulties one may encounter.

DR. P. E. TRUESDALE (closing).—In the literature I found that Sauerbruch, after experimenting with dogs, had done five resections of the esophagus in human beings, all of which were unsuccessful.

OÏDIOMYCOSIS OF THE LUNGS

Report of a Case Due to a Species of Geotrichum*

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IN A CLASSICAL study of oïdiomycosis in 1901, Ricketts[1] described three distinct types of organisms. From his description, photographs, and drawings, it is possible to identify one type as *Coccidioides immitis* (Rixford and Gilchrist[2]); one type as *Blastomyces dermatitidis* (Gilchrist[3]); and one type, which is illustrated by Ricketts, Case 3, as Geotrichum.

Coccidioides immitis infections are generally fatal and *Blastomyces dermatitidis* infections are frequently fatal, while Geotrichum infections are rarely if ever fatal. Since Ricketts' report, Geotrichum infections have been confused with those caused by the more pathogenic *Blastomyces dermatitidis*. This is the reason for reporting 5 cases of nonfatal infections with Geotrichum and giving the essential cultural characteristics necessary for the identification of this fungus.

CASE REPORT

P. L. A. No. 18409, white male, aged twenty-five years, a native of Puerto Rico, was admitted to the hospital Nov. 20, 1932, complaining of stomach trouble, cough, and expectoration for the past seven months. His tonsils were removed in June, 1932, about six months before admission. Following this operation, he developed a high fever, and the cough and expectoration increased. Roentgenograms made at that time were reported to the patient as normal, although when examined later in this hospital, they showed a dense fibrocaseous lesion in the right upper lobe. He was treated by rest in bed and with intravenous injections of some drug with which the patient was unfamiliar. He improved slowly for the next three months. After returning to Duke University in September, 1932, to continue his studies, his symptoms became exaggerated.

The physical examination on admission revealed a thin, pale, obviously ill, young man with temperature 37.4° C., pulse 100, respirations 22, and weight 57 kilograms. The general physical examination was essentially negative except for the chest, which was thin but well formed. The expansion was free and equal on the two sides. Tactile fremitus and whispered voice sounds were increased over the right upper lobe. The percussion note was dull over this area, and numerous frank, moist râles were heard on auscultation. The râles were increased in number with expiratory cough. No abnormal signs were elicited over the remainder of the right lung or over the left lung. Roentgenographic examination at the time of admission showed the gastrointestinal tract to be normal. Stereoscopic films of the chest revealed two small cavities and a mottled, fibrotic lesion in the right upper lobe and a cavity which measured 4 cm. by 2.5 cm. in the upper third of the left lung.

*Read by title at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

†From the Department of Medicine, Duke Hospital, Durham, N. C.

The sputum, which varied from 15 c.c. to 30 c.c., daily, was mucopurulent and frequently tinged with blood. It did not have a foul odor. Repeated examination of the sputum with and without concentration failed to reveal tubercle bacilli. Guinea pigs inoculated with concentrated sputum did not develop tuberculosis. Direct examination of the fresh sputum in 10 per cent sodium hydroxide showed numerous rectangular yeastlike organisms. Cultures made from bloody sputum on Sabouraud's medium gave a luxuriant growth of an oïdium-like fungus. Culture on blood agar showed a few colonies of a mucoid encapsulated organism resembling Friedländer's bacillus, and numerous colonies of the Geotrichum fungus.

Examination of the blood showed 4,800,000 red blood cells, 11,900 white blood cells, with a differential count of polymorphonuclear neutrophil leucocytes, 72.5 per cent; polymorphonuclear eosinophil leucocytes 0; polymorphonuclear basophil leucocytes, 1.5 per cent; large lymphocytes, 17 per cent; small lymphocytes, 2.5 per cent; and monocytes, 6.5 per cent. No malaria organisms were found. The blood Wassermann test was negative and the urine normal.

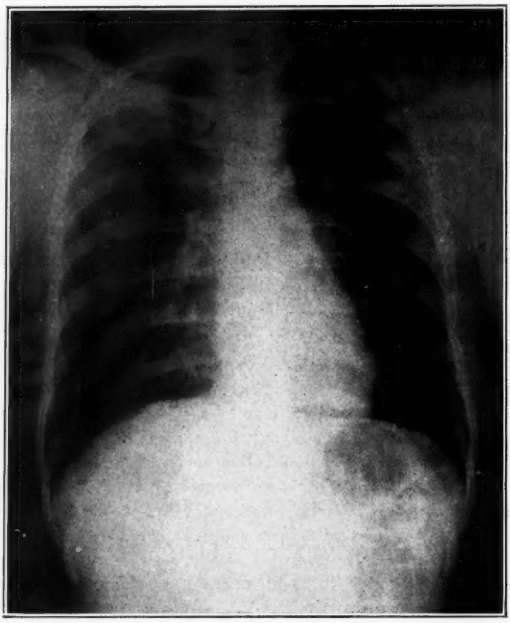

Fig. 1.—Roentgenogram of patient (Case 1) showing cavity in upper third of left lung and fibrocaseous lesion in right upper lobe.

The patient was confined to bed and fed a regular hospital diet supplemented by 15 c.c. of cod liver oil and 45 c.c. of tomato juice three times a day after meals. As soon as it was demonstrated by guinea pig inoculation that there were no tubercle bacilli in the sputum, potassium iodide was administered by mouth, beginning with 5 drops of a saturated solution three times a day and increasing gradually to 30 drops three times a day.

He began to improve within a week after admission and at the end of two weeks the sputum decreased in amount, there were fewer râles in the chest and the roentgenogram showed a reduction in the size of the cavity in the left lung and a

disappearance of the small cavities in the right lung. After two months râles could no longer be heard in the lungs, and his weight increased from 57 to 63 kilograms. On May 3, 1933, seven months after admission, the chest was normal except for slight dullness at the right apex. The roentgenogram showed some residual fibrosis at the right apex and in the center of the upper third of the left lung.

I have seen four other patients who had fungi of the Geotrichum type in their sputum.

The first case was seen in 1925 while I was working in the laboratory of the New York State Hospital for Tuberculosis, at Ray Brook, New York. The patient was a young married woman, about thirty-five years of age, who was a native of

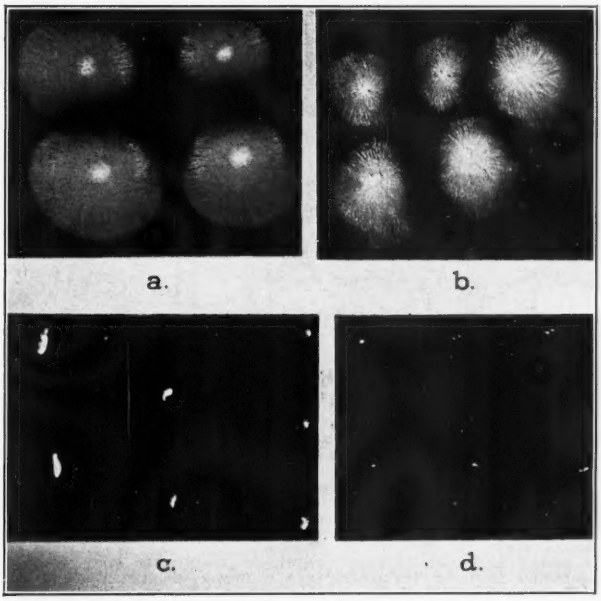

Fig. 2.—Photograph of colonies of Geotrichum after four days' incubation. Natural size. *a*, On Sabouraud's media incubated at room temperature; *b*, on blood agar incubated at room temperature; *c*, on Sabouraud's media incubated at 37° C.; *d*, on blood agar incubated at 37°C.

New York State. She presented the clinical history, symptoms, and physical findings of a case of chronic bronchitis. No parenchymatous lesion could be demonstrated by x-ray. The patient made a rapid recovery after the administration of potassium iodide.

The second case was a patient in the New York State Hospital for Incipient Tuberculosis. This patient was a young man, about thirty years of age, who presented the clinical picture of bronchitis without parenchymatous involvement of the lungs. He also improved with potassium iodide therapy.

The third patient was a native of Greenville County, South Carolina. This young girl of eighteen years had numerous fine moist râles over the posterior and lateral portion of both lungs. The roentgenogram made by Dr. W. F. Ashmore

showed numerous small soft areas of infiltration resembling tuberculosis throughout the lower two-thirds of both lungs. This patient improved with potassium iodide therapy, but was not entirely well after a year of treatment.

The fourth case was a patient of Dr. J. F. Busch of Spartanburg, South Carolina. I isolated the fungus from several specimens of sputum which he sent to this laboratory. This patient is still under treatment.

CULTURES

Patients with clinical symptoms of chronic bronchitis or pulmonary tuberculosis who have white mucoid sputum containing grayish flakes should be suspected of having an infection with yeast-like or mold-like fungi. The Geotrichum fungi grow readily on Sabouraud's media after from four to ten days' incubation. When grown at 37° C., a medium-sized, white, membranous colony appears on the surface of the media

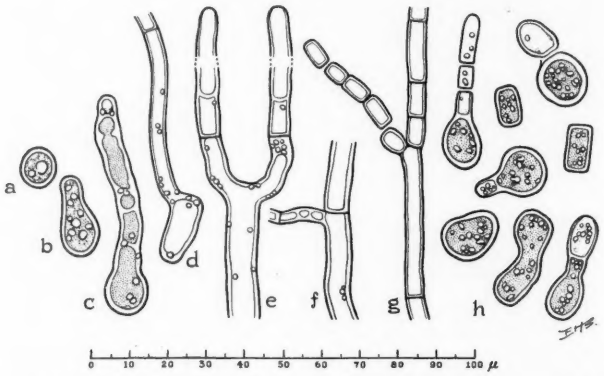

Fig. 3.—Camera lucida drawing of various stages of development of Geotrichum. *a, b, c, d,* are from an eighteen-hour surface growth on Sabouraud's media inoculated from material obtained from an old stock culture; *a* is a chlamydospore; *b* is a chlamydospore which is beginning to grow out; *c* is a chlamydospore which is growing out into a hypha; *d* is a rectangular condium which is growing out into a hypha; *e* and *f* are from a slide culture on corn meal agar after forty-eight hours' incubation at room temperature; *e* shows branching filaments with growing edge; *f* shows lateral branching; *g* is from a slide culture on corn meal agar after seventy-two hours' incubation at room temperature. It shows segmentation of hyphae with breaking up into isolated rectangular conidia; *h* is from a surface growth on corn meal agar after seven days' incubation at 37°C. Rectangular conidia, chlamydospores, and various stages of budding can be seen.

which looks tough, but is actually quite soft when picked up with the bacteriologic loop (Fig. 2). When grown at room temperature, the colony is larger and consists mainly of fine, radiating, hair-like structures which grow down into the media (Fig. 2).

The microscopic appearance varies with the type of media, temperature of incubation, and age of the culture, as shown in Fig. 3. However, the presence of rectangular conidia is diagnostic for this type of fungus, since it is the only one of the yeast-like or mold-like fungi which shows this particular shape. A detailed report of the cultural characteristics of Geotrichum is being prepared with the assistance of

Dr. F. A. Wolfe of the Department of Biology, Duke University, and will appear later in a journal of mycology.

SUMMARY

One or more species of the genus Geotrichum are apparently able to produce infection in the lungs which simulates either chronic bronchitis or chronic pulmonary tuberculosis. This organism can be cultured from the sputum on Sabouraud's media, and is identified by its rectangular conidia. This infection should not be confused with blastomycosis, which is much more pathogenic. Potassium iodide therapy is indicated.

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THE PATHOGENESIS OF BRONCHIECTASIS*

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RECENT studies have materially increased our knowledge of the various factors which enter into the pathogenesis of bronchiectasis. The interest which the Association of Thoracic Surgeons has taken in the subject of bronchiectasis has largely concerned itself with the end product of the disease, which might be remedied or alleviated by various forms of mechanical or surgical procedures.

It might be useful to a better understanding of the entire subject, to make the attempt to gather together some of the basic principles involved in the newer knowledge, in order that through a more comprehensive understanding of the development of the disease, a broader basis for intelligent preventive as well as remedial treatment may be afforded.

Our consideration of the various factors concerned in the pathogenesis of bronchiectasis will be divided into three main categories:

I. Developmental factors.
II. Disturbances of bronchial function.
III. The rôle of infection.

I. DEVELOPMENTAL FACTORS

At birth, the child is not endowed with a miniature bronchial tree and lungs, which develop proportionately in size as growth proceeds. The studies of Broman,[1] Willson,[2] Strukow[3] and Stefko[5] indicate that the permanent pulmonary parenchyma of the adult develops only after birth, and that this development is a matter of years, the minimum being three, the maximum fourteen, and the average about seven years.

At birth, the lungs of the infant consist of a very considerable proportion of interstitial tissue, transversed by the bronchi spreading out from the root of the lungs in fan-shaped manner, and terminating in the buds of the fetal bronchial tree, which buds are distended with the first postnatal breath, forming rather large alveoli in comparatively small numbers. As the lung develops after birth, new branches are formed in the peripheral part of the bronchial tree, and from these branches new alveoli are constantly developing. As this centrifugal growth continues, the original alveoli of the infant become the walls

*From the Tuberculosis Service of Bellevue Hospital, New York City.
Read at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery at Washington, D. C., May 9, 10, 11, 1933.

of the nonrespiratory bronchioles of the adult, and the peripheral bronchioles of the infant become the reinforced cartilaginous bronchi near the root of the adult lung.

As this development occurs, there is a thinning out of the interstitial framework of the lungs, and a spreading apart of the branches of the bronchial tree, so that there exists in the young infant several times as much connective interstitial tissue and bronchi to each square millimeter as exists in the lung of the adult. This infantile lung, therefore, is not a miniature copy of the adult lung, but rather corresponds more nearly to the hilum-near structures of the adult, where the periphery is as yet lacking.

Constitutional Variations.—The growth and differentiation of the lung does not occur at the same pace and in the same manner throughout all parts of the lungs in all otherwise normal persons. Recent studies by Willson,[2] Strukow,[4] and Stefko[5] indicate that individual constitutional as well as racial factors may determine the pace and manner of this lung differentiation. They show that there are individual and racial constitutional differences in the complexity of bronchial branching, the number and size of alveoli, etc. It has also been shown, as already indicated that there is great individual variation in the pace at which children grow out of the infantile lung period, varying from three to fourteen years, and it has also been shown that in some races, as in the case of the Mongols, there is a much slower and less complete development of the adult lung structure than in others, so that some persons, and many persons in some races, may retain an undifferentiated primitive infantile lung for a good many years, even up to young adult life. Such lungs have a higher percentage of interstitial tissue, a bronchial branching of less than normal complexity, and a larger proportion of these branches do not contain the normal cartilage support which keeps the lumen open, but rather are endowed with more powerful myoelastic wall structure which gives them greater mobility associated with much more extensive facilities for lymphatic drainage.

The Effect of Childhood Pulmonary Diseases.—In addition to the constitutional variations in the differentiation of lung structure, a considerable influence upon the development of the lung is exerted by the pulmonary diseases of childhood which affect the capacity of the tissues for local growth. The portions of the lungs so affected in the early infantile period are markedly influenced in the rate and character of the future differentiation, which explains many of the variations in subsequent lung differentiation between various portions of the lungs, and also establishes a predilection for these same areas for the subsequent development of disease.

The Rôle of the Developmental Factors in the Pathogenesis of Bronchiectasis.—As the terminal buds of the fetal bronchial tree distend at

birth, their lining cells separate widely, their walls are disrupted and are stretched out in form of invaginations, which thus create the air spaces into which the lining cells, soon to be destroyed, are for the most part shed.

If at any time during the prenatal period normal bronchial branching has been interfered with, then no terminal bud formation occurs; and the opening up of the lungs at birth will be opposed by bronchioles, the walls of which cannot be disrupted, nor the lining cells shed, and instead of air-space formation bronchial distention will take place. This bronchial distention may be general or local, and, accordingly, generalized or localized bronchiectasis may be produced. In the future course, the proximal end of these bronchial distentions may be stretched out, so that they become completely shut off from the bronchial trunk, losing communication with the rest of the bronchial tree. Thus, bronchiectasis develops into so-called cystic disease of the lung. Usually both, bronchiectases and cysts, occur alongside of each other, as the patency of many bronchial branches is preserved.

The extent of these anomalous structures depends upon the period at which interference with the normal bronchial branching takes place; if at a very early phase, distention of a stem bronchus occurs, resulting in the development of one or a few large cysts in the place of a normal lung lobe, or it rarely may be in place of an entire lung. Such developmental anomalies are usually associated with anomalies in other organs, and such infants usually do not survive.

Anomalies of lesser extent, however, are carried in latent form into adult life, and are usually indistinguishable from similar faulty development occurring from influences during the period of childhood.

Congenital Atelectatic Bronchiectasis.—When the respiratory function of the lung first becomes established at birth, larger or smaller lung areas may fail to open, and remain atelectatic. Such congenital atelectasis was for many years held as the main explanation for bronchiectases originating in neonatal or very early infant life. Considerable dispute still rages over this question.

The roentgen evidence from recent studies of the neonatal period indicates considerable normal inequality of pulmonary expansion in various lung areas, and also shows that infants with extensive atelectasis do not survive. It has also been demonstrated that certain lung areas which originally expand, may recollapse, so that the borderlines between strictly congenital and acquired atelectases in early infant life are obscured. But whether the air spaces were never formed, or, when formed later collapse, the future differentiation of the terminal air passages is similarly affected, and as such passages become distended, bronchiectasis results, so that every bronchial branch which does not develop into effective alveoli at birth, may dilate into a bronchiectasis; or if shut off from the remainder of the bronchial tree at

its proximal end, it may become a cystic structure. According to Tendeloo,[6] Engel,[7] and others, the lower posterior paravertebral parts of the lungs, particularly on the left side, are the sites of predilection for atelectasis in infants. It is significant that it is just these lower posterior portions of the lungs that are also the site of predilection for aspiration of dust, food particles, and inhalation infection during infancy and childhood.

Arrest of bronchial development, whether prenatal or postnatal, means the persistence of the primitive infantile type of lung from childhood into adolescence. The bronchi in such lungs do not branch normally; they become stretched and distended, and develop into bronchiectatic or cystic structures, depending upon whether they remain patent or become shut off at their proximal end.

From the above consideration of the developmental factors, it is obvious that they constitute a predisposition to bronchiectasis rather than a sole mechanism of its production.

II. DISTURBANCES OF BRONCHIAL FUNCTION

As life goes on, it is found that functional factors come to play an increasingly important rôle in the determination of the fate of bronchi, either handicapped in their development, or damaged by disease in early life. The rôle of the bronchi in pulmonary function consists mainly in bronchomotor activity and collateral respiration.

With the loss of the capacity of spontaneous and efficient bronchial contraction and distention, and the loss of the normal inspiratory and expiratory bronchial movements, there goes an inability of such bronchi to empty their contents both of the normal secretions from their mucous membrane (of the moisture normally filtered into the air passages from the blood), and of the vast amount of foreign matter constantly inhaled. Thus, the normal cleansing power of the lung is lost, and tissue fluids and secretions accumulate and stagnate in the affected air passages.

Also it has been shown that the distribution of air and the equalization of pressure within the lung are under the control of bronchomotor activity and collateral respiration. When air distribution becomes unequal because of failure of these functions, then intrathoracic traction and intrabronchial pressure exert a distending effect upon the walls of the bronchi. It is also important to realize that these disturbances of function are much more effective in infancy than in adult life. The muscular power and the rigidity of the chest wall have not yet developed in the infant, and the negative intrapleural pressure is very slight in the first few years of life. On the other hand, the mechanical forces of respiration are comparatively weaker in infancy and childhood than are the intrinsic forces of autonomous pulmonary function. The disturbance of the balance between the

extrinsic and intrinsic forces of respiratory function explains the emphysematous distention of some air spaces and the marked deflation of others, which is such a frequent occurrence in bronchopulmonary conditions of early life.

The recent work of Loeschcke,[8] and others, has emphasized the occurrence of marked inequality of air distribution throughout the lungs in bronchopneumonia in childhood, which inequality may lead to so-called pseudolobar pneumonia, or to the clinicopathologic picture which goes under the name of "bronchiolectatic emphysema." In the former, deflation is the main feature, while the latter condition represents an emphysematous distention of the preterminal and terminal bronchioles. This latter picture of "bronchiolectatic emphysema" is of interest to us in adult life, because as the bronchioles of the child pass inward relatively to become the bronchi of the adult, where they have persisted in the distended state they may subsequently show themselves in the form of the so-called "honeycomb lung." This condition is frequently described as polycystic disease of the lung, but in reality it is a condition in which the lung consists of former terminal bronchi distended in the form of emphysematous bullae.

In other instances, this "bronchiolectatic emphysema" may be only transitory, leaving, however, the sections of these bronchi in a permanently damaged state which handicaps their subsequent growth, differentiation, and future fortification which fits them to function against the more powerful mechanical forces of the adult chest.

The recent work of Schneider[16] indicates that the majority of the cases of bronchiectatic disease are preceded by a purely dilated state of the bronchi, in which the pathologic condition is simple atrophy and thinning-out of the structures of the bronchial wall. This atrophic condition is probably the basis for the later development of so-called dry bronchiectasis. Combined with the loss of self-cleansing power, it leaves these bronchi open to bacterial infection and to true bronchiectatic disease. It would appear likely that this atrophic condition described by Schneider is frequently if not usually, secondary to a previous infection in early life.

Mechanical Factors.—English clinicians, particularly, for more than two generations have been disputing over the question as to whether expiratory push (increased intrabronchial pressure), or inspiratory pressure (increased thoracic suction), is the main mechanism which distends the bronchi. Thoracic suction is the force mainly responsible for keeping a functionally incapacitated and atrophic bronchus open. Also the idea of Corrigan[9] that a cirrhotic shrinkage of the lung plays an important rôle as a dilating force upon the walls of the bronchi, has been substantiated to a considerable degree by the demonstration by Hamilton[10] that the direction of the bronchial distention coincides with that of the cirrhotic pull. This action appears also to be corrobo-

rated by the recent findings of Tendeloo[11] that bronchiectasis occurs in pulmonary cirrhosis only when centrifugal shrinkage of the connective tissue along the interlobular septa precedes that of the peribronchial tissue at a time when the bronchi are still soft and yielding. Very recently, Van Allen and Wang[20] have demonstrated experimentally that extensive fibrosis of the visceral pleura will cause marked contraction and cirrhosis of a lung lobe independent of any compression or of bronchial obstruction.

In the light of our recent knowledge of the pulmonary function this question becomes largely irrelevant, as what really matters is the functional failure of the affected bronchi. Such bronchi, having lost their control over the respiratory intrinsic and extrinsic forces, no longer possess normal control over the conditions of intrabronchial pressure, and are at the mercy of the mechanical forces active in both phases of respiration. Temporarily, a local condition may determine the predominance of either factor, but probably in all instances both factors are alternately operative.

Failure of Drainage.—Enormous quantities of fluid are poured into the lung from the blood, estimated at about 800 c.c. daily, which normally is evaporated from the lungs. In addition to this, there is a very considerable amount of bronchial mucous secretion and a great amount of inhaled foreign matter, all of which material must be taken care of if the bronchi are to function normally. The normal self-cleansing power of the lung is extraordinary. But when the bronchi become functionally incompetent, their lumen is permanently widened, and their elasticity and movements are markedly impaired. This is particularly so in the posterior inferior portion of the chest, which is also the area where the tendency for the normal moisture to gravitate is greatest, so that these lower and posterior portions become veritable gutters of the lung. As has already been shown, these same areas are the ones which are apt to be affected in developmental disturbances. The predilection for bronchiectasis of these areas is thus satisfactorily explained.

It is to be emphasized that clinical bronchiectatic disease depends upon failure of adequate drainage. Bronchial dilatation alone does not as yet constitute bronchiectatic disease. It is only when such dilatation is accompanied by failure of adequate drainage facilities for the normal secretions and inflammatory exudates, that the actual picture of disease presents itself.

Emphysema and Deflation.—With the failing drainage and the accumulation of moisture, inequality of air distribution becomes operative in the affected air passages and their tributary lung areas. Interference with the normal entrance or exit of air will occur, resulting either in emphysematous distension or collapse and edematous imbibition of other sections. In the collapsed areas, inspiratory traction

tends to distend them, while in the emphysematous areas it is expiratory pressure which exerts a similar influence.

Effect of Cough.—Another very important detrimental factor is added at this juncture to the mechanical forces already operative, namely, cough.

Cough not only serves to discharge the bronchial contents outward, but also is frequently a forcible inward air movement, which may serve the purpose of reinflating collapsed lung areas. The purpose of such a cough is to drive air into the deflated portions of the lung by way of collateral respiration from adjacent lung units. It is also obvious that such coughing must at the same time drive the bronchial contents still further downward. Before the vocal cords are burst open in cough, such a bronchus comes under the same pressure as the vocal cords themselves; and, as the air-cushioning effect of the surrounding lung is absent because of its deflation, the force of such a cough is enhanced also by the wedge-like effect of the bronchial secretions which distend the lumen of the affected bronchus.

The rôle of cough in the pathogenesis of bronchiectasis is obvious if we consider that it represents the greatest force that can arise within the lungs. Compared with the force of hard coughing, such forces as may arise in thoracic traction or in massive collapse of the lungs are insignificant. Moreover, this force is particularly exerted upon the affected bronchi by the concerted powerful contraction of the expiratory muscles, opposed by the reduction of the bronchial lumina as well as vocal cords, excepting alone the lumina of the affected bronchi. Brauer's objection that in cough the pressure is distributed throughout the lung and acts to contract the bronchi, is true for all normal bronchi freely communicating with normally inflated lung areas. But this is not the case of bronchi incapable of spontaneously reducing their lumina, and lacking the counterpressure of the corresponding lung area which is partially deflated most of the time.

Another argument against the pressure effect of cough is that it is operative for too short periods of time to produce much damage. It is evident that in the severe types of bronchiectasis, cough is very persistent, and such a great distending force, by the cumulative effect of its oft-repeated action, may produce more damage than a constantly acting but milder force.

Bronchial Obstruction.—As normal bronchi are active in both phases of respiration, bronchial function will fail equally if the bronchi are unable to discharge their air contents inward because of distal obstruction when the corresponding lung area is collapsed and indurated, or outward because of proximal bronchial obstruction. Here again, the inspiratory and expiratory forces become operative as the normal bronchial function fails, and the damaging effect of cough is obviously operative.

THE RÔLE OF INFECTION

The abnormal conditions in the bronchi, whether caused by any of the various developmental factors which have previously been discussed, or whether due to other damaging factors which disturb the functional efficiency of the bronchi, lay the basis for the actual destructive processes which later result in the form of bronchiectatic disease. These destructive influences are due to infection.

Two generations ago, Stokes,[12] famous Dublin clinician, first expressed the opinion that bronchiectasis is chiefly the result of pathologic destruction in the bronchial wall, much of which he believed oc-

Fig. 1.—*a* Acute suppurative bronchitis in a child.

curred during childhood. Recent extensive studies by Ireland,[13] McNeil[14] and others, as well as Erb,[15] have served to emphasize Stokes' concept of the underlying pathology of the disease. The estimation of the infectious-pathologic factors may be considered in two distinct periods: first, that of childhood with its characteristic bronchopneumonic and interstitial processes, and, second, the period of actual bronchiectatic disease with its characteristic processes of destruction of the bronchial walls.

The Pathologic Conditions of Childhood.—As was pointed out earlier, the lungs of infants and children, being imperfectly developed, possess a far greater proportion of interstitial framework and a greater proportion of bronchi to unit space of tissue than does the adult lung;

and, associated with this, there is a proportionately greater amount of lymphatic channels and lymphoid structures. The result of this anatomic difference is that in the presence of pulmonary infection in adults there is a larger proportion of exudate into the alveoli, which can absorb completely after the inflammation has subsided. During childhood, on the contrary, the interstitial framework and its extensive interlobular and peribronchial tissues are more frequently invaded, and associated with this the lymphatic channels and neighboring lymph nodes are severely involved, blocking lymphatic drainage and frequently interfering with the free intake of air into the bronchi

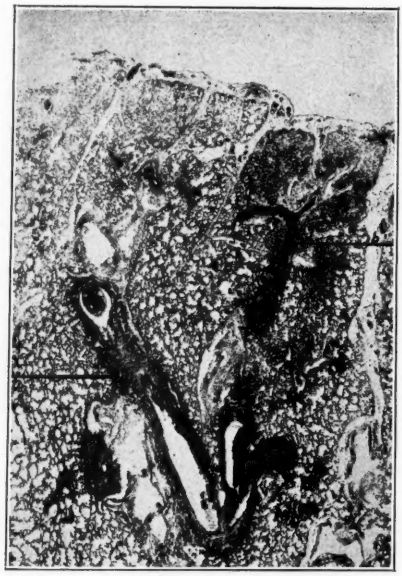

Fig. 2.—Subacute suppurative bronchitis in a child; *a* inflammation of the wall of the bronchus; *b* extension into the surrounding parenchyma.

more severely involved. Such conditions resolve with much greater difficulty, and consequently pulmonary infections in childhood much more frequently result in chronic, more or less permanent, pulmonary sequelae. According to recent studies, the types of infection which most likely result in these chronic changes, are the influenzal, the mixed influenzal and streptococcic, and the bronchopneumonias associated with measles and pertussis.

Considering the great frequency of such pulmonary infections in childhood, it is astonishing that their chronic sequelae resulting in bronchiectatic disease are not more frequently found in clinical practice. This probably can be explained by the fact that the lungs of children are still in their developmental phase, and by virtue of their

continued growth, they may be capable of restitution and elimination of destructive processes which on first impression might seem irreparable.

McNeil and his coworkers, and more lately Erb, as well as others, have demonstrated that adult bronchiectatic disease often represents the direct sequelae of the bronchial destruction suffered during infancy and childhood. In the words of McNeil and his associates, they conclude that: "The bronchiectatic cavity is not a dilated bronchus, but an excavation in the lung substance, starting in a bronchus. . . . . It is not necessary in order to explain the existence of the cavities to

Fig. 3.—Subacute suppurative bronchitis in a child; *a* productive inflammation of the bronchial wall, with extension into the surrounding parenchyma and bronchovascular connective tissue sheaths; *b* extension into the septa.

postulate the operation of any of the factors usually credited with dilating effects. Destruction of tissue and not dilatation is the essence of the process." This proposition is supported by Erb's very recent studies describing the characters of these lesions in the chronic state of incomplete repair, namely, the dilated bronchus consisting of granulation tissue which has practically replaced all of the normal structures of the bronchial wall which however may become reepithelized. Thus, it apparently cannot be denied that much bronchiectases of older children and young adults represent the direct results of bronchial destruction following a state of incomplete repair to damage sustained in childhood. As already indicated, this early damage

results in defective growth and the development of a type of bronchi which will ultimately succumb under functional stress, and which is particularly susceptible to later bacterial infections.

In Fig. 1 the lesions *a* of an acute suppurative bronchitis in a child are shown. Such lesions may become absorbed, but the reparative process would leave a bronchus with impaired function, possibly predisposed to true bronchiectasis in later life.

In Figs. 2 and 3, the lesions of a subacute suppurative bronchitis in a child are shown, with beginning productive inflammatory changes involving the wall of the bronchus, with *a*, extension into the surrounding parenchyma and the bronchovascular connective tissue sheaths, and, *b*, extension into the septa.

Such damaged tissue carried into adult life undoubtedly predisposes to later bronchiectatic disease.

The Pathologic Process in Bronchiectatic Disease.—There is, however, much evidence in support of the contention that the great bulk of adult bronchiectases are not the direct sequel of childhood bronchial destruction, that other superimposed conditions are necessary to complete the picture. Schneider[16] has recently given us a most extensive, illuminating series of studies on the pathology of bronchiectatic lesions.

The two main points which he emphasizes are: (1) that in every bronchiectatic lung with lesions, no matter how extensive, there can always be demonstrated adjacent bronchi in the purely atrophic state of bronchial dilatation, and (2) that there can always be found bronchi showing the first phase of the infectious pathologic lesions, and that these are found in the subepithelial layer of the bronchial wall under the intact mucous membrane, as well as over intact subjacent structures of the bronchial wall.

From these two observations Schneider brilliantly reconstructed the sequence of events in the infectious pathologic process which constitutes the bronchiectatic disease, as follows: That there is first a purely atrophic, cylindrical bronchiectasis, that this is followed by an atrophic bronchiectasis with chronic inflammatory changes in the subepithelial layer, which lead to cellular infiltration of the bronchial wall, which he terms hyperplastic bronchiectasis. That the final end-result is an organization of this hyperplastic tissue into fibrous tissue, developing what he calls cirrhotic bronchiectases, and with the consequent loss of elasticity of the wall saccular bronchiectases, which latter he divides into those with elastic and others with rigid cavity walls.

Schneider briefly summarizes his pathologic conclusions as follows:

"One must distinguish between atrophic and hyperplastic bronchiectases. The first are cylindrical, and the latter include both cylindrical as well as saccular bronchiectases. The inflammatory process in hyperplastic bronchiectasis is a purely subepithelial one, and eventually leads to the destruction of the entire wall.

"The purely atrophic bronchiectases arise by increased intrabronchial pressure due to involvement, and exclusion from function, of other adjacent lung areas. These may go over into the hyperplastic bronchiectases.

"The hyperplastic or so-called inflammatory bronchiectasis develops by way of infection of the bronchi, frequently in connection with transitory pneumonic processes, but chronic pulmonary processes as such do not play a definite rôle in its evolution. Whether purely atrophic processes in the lungs, without mechanical factors, and without inflammatory processes in the wall of the bronchi, may lead to

Fig. 4.—Early bronchiectasis of left lower lobe in an adult dying from other causes. Proliferative inflammatory process extending from bronchi into bronchovascular connective tissue sheaths and septa. A general view of this section is shown, with lesions at *a* and *b*, as well as other areas.

diffuse or circumscribed bronchiectasis, must remain an open question."

As we have already noted, it is probable that these atrophic processes described by Schneider may be the remains of previous inflammatory changes, perhaps dating back to childhood.

According to Schneider, the inflammatory with their subsequent cirrhotic processes do not spread into the adjacent lung tissue until late in the disease. In the atrophic and hyperplastic stages, the adjacent and tributary lung areas are usually found in hyperinflation and some in hypoinflation. It is during the cirrhotic phase that inflammatory changes frequently invade the pulmonary parenchyma, and from

then on evidence of repeated bronchopneumonic processes may be found. Such bronchopneumonic processes may then extend to the pleura, often ending with permanent adhesions and induration of all the surrounding lung and pleural structures. Such processes may appear exactly as though primary pleuropulmonary lesions had invaded the bronchi and produced the cirrhotic induration of their walls, with the bronchiectases as the secondary result instead of the primary disease.

As a matter of fact, as already indicated in our discussion of the mechanical factors, such processes may extend from the pleura and the lungs into the bronchi. But extension of the process in the reversed direction seems to be the more usual one. In bronchiectases associated with chronic infections of the lung, such as tuberculosis, pulmonary abscess, fibrosis, pneumoconioses, etc., the formation of the bronchiectases may be due to disturbances in the bronchi, or they may result secondarily to the interstitial changes in the lung and pleural structures themselves. A considerable amount of confusion exists in the literature concerning the interaction of interstitial processes in the lung and associated bronchiectatic disease. But recent developments of our knowledge favor the primary influence of the bronchial factors.

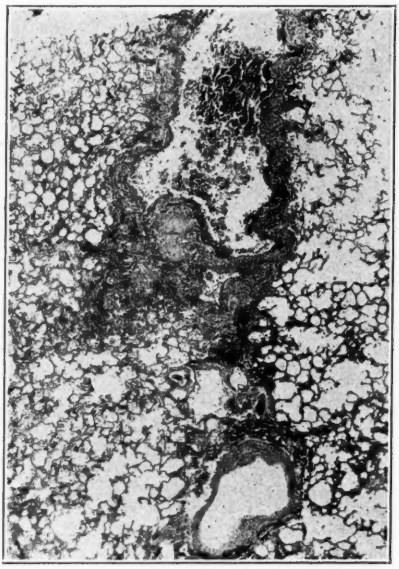

Fig. 5.—Same case. Showing enlargement of the lesion *a* of Fig. 4.

In Figs. 4 to 9 are illustrated various lesions in a case of early bronchiectasis in which the patient died from other causes.

Fig. 6.—Same case. Showing enlargement of the lesion *b* in Fig. 4.

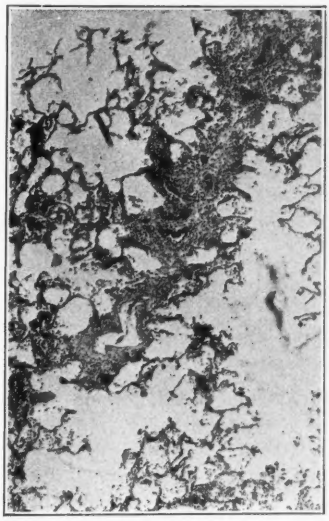

Fig. 7.—Same case. Showing productive-inflammatory process in the interlobular septum.

Fig. 4 shows a general view, with special lesions at *a* and *b* as well as in other areas, showing the productive inflammation involving the walls of the bronchi and extending into the surrounding tissue. The details of these processes are shown in enlarged view in Figs. 5 and 6.

In Fig. 7 is shown the involvement of the interlobular septum by extension from the bronchus. It is the involvement of these septa, according to Tendeloo, that may cause dilatation of the walls of the bronchi by traction, especially in children when the walls of the bronchi are soft and elastic.

Fig. 8.—Same case. Showing in other areas the productive-inflammatory process extending from the bronchus into the adjacent bronchovascular connective tissue sheaths and septa.

In Fig. 8 more marked extension of the process from the bronchi into the parenchyma is seen, and the same section is shown enlarged in Fig 9 in greater detail.

The Influence of Bronchial Obstruction.—In the pathogenesis of bronchiectasis associated with extensive pulmonary cirrhosis, we must attribute a significant rôle to bronchial obstruction. Erb in his recent pathologic studies called particular attention to mechanical obstruction, which he believes to be a most important factor in every form of bronchiectasis. Van Allen[19] in his recent studies on collateral respiration in the lung, has taught us that mechanical obstruction is effective only if it involves one of the stem bronchi of the five lobes of the lung. This would confine the rôle of bronchial obstruction to the so-

called atelectatic bronchiectases involving a lobe or an entire lung. An exception to this must be in those cases, more numerous than is usually appreciated, in which there are congenital abnormalities in the lung resulting in the accessory lobe. In such cases, obstruction of the bronchus leading to such an accessory lobe would act in exactly the same manner as that of a lobe bronchus, inasmuch as there is no collateral respiration between such lobes. The importance of these accessory lobes has recently been emphasized by Soper.[17]

The usual causes of bronchial obstruction are various forms of enlargement of the tracheobronchial glands, whether they be tubercu-

Fig. 9.—Same case. Enlargement of a portion of section shown in Fig. 8, showing the details of the extension of the bronchial lesion into the adjacent bronchovascular connective tissue sheaths.

lous or acutely inflammatory, or anthracotic; foreign bodies, and the inhalation of small foreign bodies, such as particles of food, is important, especially in children; neoplasms, both malignant and benign, aortic aneurysm, etc. After mechanical obstruction from whatever cause occurs, the rôle of infection becomes particularly damaging, and in such cases the destructive processes go on even more rapidly than in cases in which no bronchial obstruction exists.

The Influence of the Nature of the Bacterial Infection.—Whether the destructive action of bacterial infection operates in a functionally damaged bronchus or in an obstructed bronchus, the destructive processes are due to the infection. Recent studies of Greey[18] indicate that

any of the ordinary pyogenic organisms of respiratory disease may produce this effect. That certain anaerobic organisms, either alone or in combination, may cause similar or even excessively destructive processes, is well known. Particularly this is true in those cases associated with bronchial obstruction. The work of Greey, however, seems to point very definitely against the contention of David Smith[21] and others, that anaerobic infection is the essential factor in the production of bronchiectasis. Greey found such organisms in considerable numbers only once in a series of nine carefully studied cases following lobectomy. This evidence contradicts the suggestion that bronchiectatic disease should be designated a fusospirochetal disease, a point which has its important bearing not only on the etiology but also on the treatment of this condition.

The Influence of Associated Infections in Upper Air Passages.—When we consider the opportunities for infection in the bronchi, whether previously damaged or not, we cannot omit consideration of chronic associated infections in the upper respiratory tract. That patients who have chronic infections of the sinuses, tonsils, teeth, are susceptible to recurrent attacks of bronchitis and bronchopneumonia, is well known. If such infections occur in patients who, for some of the various reasons previously discussed, have already damaged or functionally inefficient bronchi, the likelihood of the development of true bronchiectatic disease is of course materially enhanced. Moreover, the prevention of such infection of the bronchi would depend upon the elimination of the original sources of infection.

Erb in discussing this feature of the problem, suggests that the process of secondary infection may sometimes be reversed, and that sinus infection may be the result of the constant expectoration of large quantities of foul sputum from bronchiectatic cavities.

Whatever may be the specific nature of the bacterial infection or its source, all recent evidence clearly points to the fact that a combination of functional and infectious pathologic processes is essential for the evolution of true bronchiectatic disease.

DISCUSSION AND SUMMARY

Recent studies concerning the pathogenesis of bronchiectasis would therefore lead us to the conclusion that there may be certain developmental abnormalities which predispose toward the later development of true bronchiectatic disease, that however it is extremely difficult to differentiate between changes which are strictly congenital and those which may take place in the early years of life as the result of postnatal disease.

We have further seen that the fate of these bronchi, whether handicapped in their development or damaged by disease in early life, is

largely determined by certain functional factors, and that the most important of these are bronchomotor activity and collateral respiration. The disturbances of either or both of these important functions of the bronchi, which may be associated with bronchial obstruction, inadequate drainage, atelectasis, emphysema, or pulmonary cirrhosis, lay the groundwork for the action of bacterial infection, which is the actual cause of true bronchiectatic disease.

These bacterial infections may act either in early childhood, causing disturbances of normal development of the bronchi, or, as is more important and more usual, they exert their influence by destructive processes in the walls of the adult bronchi.

Just as the predisposition to bacterial infection depends upon developmental and functional factors varying in different individuals, so also the opportunities for repeated infections vary greatly in different individuals, particularly in regard to associated infections of the upper air passages. It is also seen that anaerobic infection is by no means the essential factor, that any of the ordinary bacteria of acute respiratory disease may act in the same fashion.

To those of us who have become accustomed to think of bronchiectatic disease in its more terminal phases, it is instructive to trace the path along which this disease gradually develops; and to the internist particularly, it offers the hopeful prospect of counteracting the various unfavorable influences in their earlier stages, and thus to hope for the prevention of much of the bronchiectatic disease as it exists today. Such efforts at prevention would be directed particularly to the care of the apparently slight pulmonary affections during the early years of life, with their far-reaching possibilities, and in the adult it would point to the importance of eliminating as far as possible sources of infection in the upper air passages before much damage has occurred in the bronchi themselves.

From the standpoint of treatment, a clear idea of the developmental factors would clarify our notions concerning such widely disputed problems as those of congenital cystic lung, honeycomb lung, etc., and bring these into line with the other forms of bronchiectatic disease. An appreciation of the gradual influence of the failure of bronchial function in the development of bronchiectatic disease would repay the study of internists and surgeons alike, in clearing up that uncertain borderline zone which lies between a chronic bronchitis and a well-developed bronchiectatic disease.

Appreciation that failure of drainage is one of the most important factors would tend to confirm our present surgical notions that only radical elimination of the foci of disease will effectively cure well-established, advanced bronchiectasis.

Our experiences with less radical measures, such as artificial pneumothorax and phrenicectomy, have not been satisfactory. These measures in our hands have quite frequently resulted in blocking of drainage and in exacerbation of symptoms.

As internists and surgeons together conduct further studies in this borderline zone of cases, an appreciation of the predisposing developmental and functional factors would be an aid in the prevention of some cases, and in others more satisfactory treatment of pulmonary affections during infancy and early childhood would also be repaid by the prevention of many cases of bronchiectatic disease as it now exists. From the standpoint of the surgeon, improving technic should render it possible gradually to extend radical and curative procedures back to those less advanced cases which now are allowed to drift into well-developed cases. So that working hand in hand, our patients would benefit from either more effective preventive management, or, if need be, more effective and safe radical surgical treatment.

I wish to acknowledge my deep indebtedness to Dr. Wolfgang Grethmann, pathologist of our Service, for most valuable assistance in the preparation of the pathologic material; and to Dr. Charles Hendee Smith who has courteously allowed me to use the pathologic material of his Service in showing examples of infection in childhood; and especially to Dr. Israel Rappaport for most valuable assistance in the study of the literature and the arrangement of the data presented.

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DISCUSSION

DR. CARL A. HEDBLOM (CHICAGO).—Dr. Miller's observations on the development of bronchiectasis seem to me of great importance with respect to prophylactic treatment. In my opinion two factors contribute to the pathogenesis of the ordinary type in which there is no definite evidence of bronchial obstruction: namely, infection that weakens the bronchial wall and mechanical force represented by the difference between intrabronchial and extrabronchial pressure. We can perhaps do something toward prevention of intrabronchial infection, particularly that incident to the diseases of childhood, and we can do much toward checking the process of dilatation in the early stage by partial equalization of the pressures by phrenic nerve crushing or extraction and in some cases by therapeutic pneumothorax. Relaxing the lung in the presence of extensive fibrosis will serve a similar purpose.

Regarding the mechanism of intrabronchial dilatation it would seem to me that it must be the inspiratory phase of coughing which increases to a maximum the difference between intrabronchial and intrapleural pressure that produces the dilatation, rather than the expiratory phase during which the intrapleural pressure most closely approaches that inside the bronchi. In pulmonary atelectasis the intrapleural pressure may be reduced to 50 to 100 cm. less than atmospheric pressure. Under such conditions, that measures the dilating force of intrabronchial atmospheric pressure.

In the early stages of bronchiectasis phrenic neurectomy in my experience has produced symptomatic cures, persisting without relapse for a period of several years, and lipiodol bronchography in such cases has failed to demonstrate bronchial dilatation.

DR. HOWARD LILIENTHAL (NEW YORK CITY).—While I fully agree with Dr. Hedblom's treatment, I do not agree, if I understood him rightly, with his explanation of the dilatation of the bronchi.

It seems to me that the bronchi are forcibly dilated when cough occurs, because with the larynx closed at the beginning of the effort there is a tremendous pressure from the healthy side into that of the disease.

DR. JOHN ALEXANDER (ANN ARBOR).—My experience confirms what Dr. Hedblom has just said about phrenic paralysis. I have had as patients a number of children from five to fourteen years of age, as well as older persons, with suppurative pneumonitis of the type that I believe corresponds with that demonstrated by Dr. Miller; for them I have performed a temporary interruption of the phrenic nerve.

When phrenic paralysis is induced in the early stages of an infiltrative lesion whose course indicates "delayed resolution," but not abscess formation or chronic bronchiectasis, I have been greatly pleased with the results and now use the operation almost routinely. The phrenic paralysis obviously gives far greater rest to the diseased tissues than does bed rest alone. It lessens the respiratory tug upon the weak and infiltrated areas of the lung which are potentially bronchiectatic. It tends greatly to shorten the period of months or years during which, if collapse therapy

is not used, some children should be kept quiet and away from school because of recurrent febrile attacks, persisting physical signs of the lesion and cough, with or without purulent sputum.

My experience has been that the five or six months of hemidiaphragmatic paralysis that a temporary interruption of the phrenic nerve gives is usually sufficient to cause resolution of young lesions and so I prefer a temporary to a permanent interruption. Although an induced pneumothorax would produce more pulmonary rest and relaxation than a phrenic paralysis, I believe that the latter is sufficient in most cases, and it avoids the dangers of a pneumothorax and particularly the danger of empyema to which pneumothorax patients with subacute pulmonary suppurative disease are especially exposed.

POL. N. CORYLLOS (NEW YORK).—Dr. Miller presented to us today a brilliant paper on bronchiectasis. Yet, I do not think that he brought us any definite solution of the most puzzling part of this disease, which is its pathogenesis. I shall limit myself to the discussion of that point.

Dr. Miller has stressed the point that the most different pulmonary lesions, mechanical (bronchial obstruction by foreign bodies or tumors, stenosis or compression of the bronchi or compression of the lung as in chronic empyema or thoracoplasty) or infectious (chronic bronchitis, bronchopneumonia, pneumonia, measles, whooping cough, tuberculosis, abscess, chronic sinusitis, etc.) may lead to bronchiectasis. Practically every pulmonary disease can produce bronchiectasis.

On the other hand in the slides projected it was clearly shown that the bronchiectatic lung is fibrotic and atelectatic, with occasional spots of emphysema interposed. The dilated bronchi show advanced lesions of their musculoelastic element. In the x-rays, with lipiodal injection, and especially in the saccular forms, we see very clearly these dilatations, but we see no fine bronchi, bronchioli, and alveoli injected, as in the healthy parts of the lungs.

These are the facts. Is it possible to correlate them and arrive at a rational explanation of the production of bronchiectasis?

I think we can. In fact every one of the above mentioned diseases is liable to produce obstructive or compressive atelectasis, lobar or partial. If the causative factor is removed early, the lung will be reaerated and become normal. On the other hand, if the cause persists, an acute or chronic infection will follow, producing an acute or chronic pneumonitis, according to the nature, number, and virulence of the microbes involved. In chronic infections, or infections which become chronic there invariably occurs what Letulle has called "plastic alveolitis"; that is, organization of the exudate and closure of the alveoli of the regions involved. This "definite atelectasis" will be followed by development of fibrous tissue thus causing permanent shrinkage and closure of the alveoli. The alveoli which have escaped infection and closure will present, on the contrary, vicarious emphysema.

Here is, I think, the connecting link between all the above mentioned pulmonary diseases and bronchiectasis.

From now on the events will proceed in a way which is in perfect accordance with the clinical course of the disease.

Closure of the alveoli will deprive the affected portion of the lung of its most powerful means of drainage, namely, the draft of air during respiration and especially during cough. In the bronchi terminating in the culdesacs, like test tubes, stasis of more or less infected bronchial exudate will naturally follow. This phase corresponds to the "chronic bronchitis" with cough and more or less expectoration. Any acute affection of the upper respiratory ways may impair drainage because of the edema, infectious or possibly allergic in origin of the bronchial mucosa, which

often follows these infections; Graham has well shown this fact in bronchial fistulas. This explains the acute episodes in the course of these so-called "chronic bronchitides."

This prolonged infection affects the musculoelastic element of the respective bronchi, and decreases the resistance of their walls. Furthermore oxygen will be absorbed by the aerobic microbes and the cells present in the exudate, while renewal by the respiratory air or the circulating blood is diminished, because of the atelectatic shrinkage of the parenchyma. This change of the medium will allow the development first of facultative and later strict anaerobes. Development of anaerobes will enhance the destruction of the musculoelastic element of the bronchial wall thus decreasing its resistance; above the affected portion of the bronchus, on the contrary, the lumen will be narrowed because of the spastic contraction of the healthy smooth bronchial muscles which is naturally far more pronounced in the living individual than is seen in the postmortem specimen. This point is of great importance as will be shown later.

Thus the clinical picture of bronchiectasis is gradually established.

The second point of importance is the mechanism of production of bronchial dilatations. If I am not mistaken, three opinions were supported here today; the old opinion of Kiernan of traction exerted upon the softened bronchial walls by the retracting connective fibrotic tissue; the action of the increased negative intrapleural pressure, and the action of cough.

It is time, I believe, to drop the first theory. Charcot had already proved its weakness. It is irrational to claim that the same connective tissue in the same lobe will on one hand dilate the bronchi and on the other hand squeeze the much less resistant alveoli. The same argument holds against the second theory.

It is not the same with cough. The conception advanced by Dr. Hedblom that during cough the intrapulmonary pressure is the same everywhere is unwarranted; it considers cough as a whole and disregards the fact that cough is composed of three separate phases greatly different, mechanically speaking, from each other.

In the first or the "inspiratory" phase, the lungs are filled with air. During the second phase, which I shall call the "compressive," the chest performs the act of forced expiration *while the glottis* is tightly closed; thus the air is compressed in the lung, up to 60-80 mm. of Hg, and in that phase of cough the pressure is equal everywhere in the lungs. In the third phase, which I shall designate as the "explosive" phase, the respiratory contraction of the chest persists, but the *glottis is slightly opened,* allowing a strong draft of air to be expelled carrying with it mucus and other foreign bodies present in the bronchi. Cough can thus be compared to the explosion of a cartridge in a gun, with its three distinct phases of loading, conflagration of powder, and expulsion of the bullet.

It is during this third period, I believe, that bronchial dilatation is gradually produced. In fact, at the opening of the glottis the compressed air is readily expelled from the healthy portion of the lung through the wide open normal bronchi. It is not the same in the diseased areas; because of the constriction present above the diseased segment of the bronchi the air exit will be much slower; therefore, whereas the pressure in the normal lung falls readily to zero, in the diseased portion it falls much slower and often the following inspiration finds the diseased bronchi still under positive pressure. In that way during the third phase of cough the pressure inside of the diseased bronchi becomes higher than in the surrounding healthy parenchyma; thus the softened bronchial walls gradually yield, uniformly at the beginning, when the lesions are uniformly distributed (cylindrical dilatation) and irregularly in more advanced stages of the diseases where portions of the walls show more advanced lesions than others (saccular and moniliform dilatations).

The bronchi are thus dilated under the influence of cough exactly as a glass tube softened over a flame is blown at the heated areas when the air pressure into it is raised.

The succession of these phenomena is not theoretical and speculative; they can be shown very clearly in a model constructed in my laboratory at the department of Surgical Research of Cornell Medical College. I shall present it to you at our next meeting.

DR. E. J. O'BRIEN (DETROIT).—I just want to corroborate Dr. Hedblom's experience with phrenicectomy in early bronchiectasis. There are those who state that phrenicectomy is of no value in this disease, and that lobectomy should be performed. They, no doubt, base their opinions on results of phrenic nerve operations in an advanced stage of the disease. It is difficult for me to understand just why some persons cannot or will not see that the results from these operations will depend almost entirely upon the stage of disease for which they are applied.

There is a great similarity between the treatment of pulmonary tuberculosis and bronchiectasis. In tuberculosis, the results from phrenic operation will likewise be best in patients with early minimal disease, but here again, many men refrain from using the operation until the disease is far advanced. Then because no result is obtained, the procedure is blamed.

DR. C. M. VAN ALLEN (PEIPING, CHINA).—The acute necrotizing lesions of the bronchioles which Dr. Miller has shown are particularly interesting to me. He apparently believes them to be the forerunners of bronchiectasis, in that, after healing, they leave the bronchiolar walls weakened at those points and incapable of resisting the dilating influence of cough. It seems to me very unlikely the bronchiectasis forms at the sites of these lesions, from what we know of the manner of healing of acute necrotizing lesions of the bronchial walls in experimental animals. Dr. W. E. Adams and others have found that when the wall of a bronchus is damaged by chemical or thermal agents to the extent of devitalizing the mucous membrane and the supporting elements (cartilages), during the healing process that ensues cicatricial contraction and stenosis result regularly; and when the necrosis is made to encircle the bronchus, the stenosis becomes complete with total occlusion.

If these lesions of the small bronchi pictured by Dr. Miller are ever the forerunners of bronchiectasis, the process would seem more logically to be the following: After healing, the bronchioles are closed at the sites of the lesions, and the portions of the tubes remaining patent on the proximal side, up to the nearest bronchiolar branch, are "blind" passages disconnected from the alveoli which they formerly supplied and therefore devoid of aerodynamic protection against obstruction which normal bronchioles possess. Those situated in the lower parts of the lungs have their open, proximal ends directed upward, so that they tend to receive and hold gravitating secretions. The stagnation of fluid in these tubes conduces to chronic infection of their walls, which in turn tends toward gradual degenerative changes with loss of the elastic elements of those tissues. The loss of elasticity leaves the tubes unable to resist for long the broncho-dilating force of cough. I present this hypothesis without conviction, even though it is supported by the fact, just brought forward by Dr. Coryllos, that lipiodol introduced into bronchiectatic bronchi is shown by x-rays to stop short of the periphery of the lungs and not to reach the alveoli, as it often does when introduced into normal bronchi. This stoppage, of course, does not mean necessarily that the bronchi are stenosed, for the lipiodol may be blocked merely by thick exudate. Although without secure support, the hypothesis is the most likely known to me.

I certainly cannot agree with Dr. Coryllos in his claim that partial stenosis of the bronchus proximal to the bronchiectatic dilatation is an essential part of the

process; for I have seen many bronchograms showing complete patency of the bronchi in point. Also, I cannot agree with him that atelectasis plays an essential rôle in the pathogenesis of bronchiectasis; because roentgenograms in that disease frequently show no consolidation of the parenchyma distal to the dilated bronchioles. The lack of atelectasis does not militate against the hypothesis I presented, since we know that complete occlusion of bronchioles does not necessarily lead to atelectasis of the lobules supplied by them. The lobules of occluded bronchioles may remain air-containing for indefinitely long periods of time on account of collateral respiration through the adjacent lobules possessing patent bronchioles.

DR. JAMES ALEXANDER MILLER (closing).—There are only one or two points in the discussion which I wish to take up; in the first place concerning the question of cough. We could probably discuss the theoretical effect of cough indefinitely without reaching an accurate conclusion. But I believe that it is undoubtedly true that both the inspiratory and expiratory phases of respiration have their influence. Brauer held that cough could not have the effect of dilating a bronchus, because the pressure was equalized on all sides. But what we wish to emphasize is the fact that the wall of a bronchiectatic bronchus is weakened, and that in such case the effect of cough upon that particular bronchus may be very considerable, because it is not equally supported by normal parenchyma about it.

As to atelectasis, no doubt, as in Dr. Coryllos' case, it is a very important factor when bronchial obstruction is present. But I am not at all convinced that atelectasis is the only or even the chief factor in the usual causation of bronchiectasis.

As to treatment, although I am convinced from my own experience that artificial pneumothorax and phrenicectomy will often do harm in advanced cases of bronchiectasis, the experience of others, particularly Rist, may lead one to hope that in early cases these measures may be of value. I must confess that I have not personally given them sufficient trial in early cases to be sure of this point.

Finally, in connection with Dr. Van Allen's discussion I think he obtained an incorrect impression which I trust will be made clear when the full paper appears in published form. It is not my belief, as Dr. Van Allen apparently thinks, that the effect of the peribronchial fibrosis is to dilate the bronchus, but rather that the infection of the wall of the bronchus is the main factor, weakening and undermining the elastic structures.

The object of this presentation has been achieved if the attention has been directed to the importance of early and slight lesions, particularly infections, which predispose to what we now term bronchiectatic disease, and that as internists our responsibility for prevention is probably greater than is the responsibility of surgeons for the radical cure of advanced bronchiectasis.

THE EXPLORATION OF THE LESION AND PNEUMO-CAVERNOLYSIS IN THE OPERATIVE TREATMENT OF PULMONARY TUBERCULOSIS*†

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THE earlier theories on which the surgery of pulmonary tuberculosis was based invoked the principles of relaxation, immobilization, and compression of the diseased lung. Healing was thought to have been invited by thoracoplasty as the mechanical means of application of these principles. In recent years there has gradually developed a realization that the essential objective of the operative treatment of pulmonary tuberculosis should be the collapse of cavities. That the customary thoracoplasty may achieve this purpose is universally accepted. However, a reading of the literature and personal experiences have convinced me that thoracoplasty is successful only so far as collapse of cavities is the result. Since thoracoplasty is an indirect, blind approach to the problem, the outcome is uncertain in any given case. A patient may be subjected to the excision of the entire hemithorax and still be left with a stiff-walled uncollapsed cavity.

Another defect of the standard operation, inherent in the nature of the procedure in my opinion, is its morbidity and mortality. Regardless of the collapse and immobilization which may be ultimately attained, the immediate mechanical effect is the release of the lung from its moorings to the chest wall and the resultant agitation of underlying tuberculous lesions. Only a considerable fibrosis will reduce the mobility invited by thoracoplasty, so that a widespread tuberculous seeding is all too often the direct sequel of operation.

The voluminous literature dealing with efforts at more direct compression of the tuberculous lesion is evidence of dissatisfaction with the standard operation. These efforts are on the one hand concerned with selective thoracoplasty, and on the other, with extrapleural release of approximately normal lung in the region of the tuberculous process (apicolysis, pneumolysis). Less blind than the standard operation they nevertheless depend for their efficacy on the degree to which the cavity can be collapsed by indirect means. They can succeed only so far as such collapsibility exists, and the outcome of methods of indirect collapse cannot therefore be accurately foretold.

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A strange aspect of the operative treatment of pulmonary tuberculosis, in striking contrast to surgery in other fields, has been an unwillingness to investigate the pathologic process at operation. The main purpose of this presentation is to describe a procedure based on the operative exposure and investigation of the site of cavitation, as developed in the past two years. The results which have been obtained can be referred to only tentatively at this time and variations in the operative steps which are at present contemplated may appreciably alter the results in future cases. It is the principle of the exposure and investigation of the lesion and the application of suitable measures in accordance with the findings which I wish to stress.

Brief reference should be made to the obvious necessity which existed for obtaining precise information of the situation and extent of the cavernous lesion, with its frequently associated fibrosis and bronchiectasis, before proper direct operative treatment could be evolved. Bronchography was employed for this purpose. The results have been reported elsewhere.* It can be stated here that selective bronchography has been practiced in more than a hundred cases at our institution without untoward results and with important knowledge gained concerning: (1) the site of cavitation; (2) the differentiation between cavity and bronchiectasis; (3) the site and extent of bronchiectasis, with its inferential evidence of fibrosis; (4) the status of the bronchial tree in other lobes; (5) the effect of operation on the cavity and adjoining bronchial tree; (6) the reasons for failure or incomplete collapse after operation. The information derived from bronchographic study has had a definite bearing on the extent of operative exploration and efforts at collapse in the operation to be described.

Tuberculous cavitation was confined to the upper lobe in most of the patients subjected to operation and the description of operation will therefore be limited to upper lobe lesions. The scapula is mobilized and lifted from the chest wall by an incision beginning opposite its spine and carried downward around and well in front of its inferior angle. The approach to the lesion is obtained by subperiosteal costectomy of several of the upper ribs, usually the first four to six. The number removed and the site and extent of excision depend on the size and position of the lesion. The intercostal tissues and adjacent periosteum, which are sometimes infiltrated and adherent to the underlying pleura, are dissected free and reflected mesially in the form of flaps. The cavity is usually situated near the surface, only a thin shell of lung ordinarily separating its roof from the visceral pleura. The lesion can therefore be palpated, and seen to a varying extent, after reflexion of the musculoperiosteal flaps. It may prove to be soft

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and collapsible by direct pressure or by pressure toward the mediastinum applied against the lateral stumps of the cut ribs. Collapse may be aided by extrapleural stripping around the site of the cavity. The pleural cleft was diffusely sealed by adhesions over the lesion in all the cases. Thin adhesions are no bar to the collapse of the cavity. When there is pronounced thickening of the pleurae, however, adequate collapse of the cavity does not occur with the simple manipulations which have been mentioned. No alteration in size or shape will be noted in the old, stiff-walled lesions. The step I have termed "cavernolysis" is carried out if inadequate or no collapse occurs because of the thickened agglutinated pleurae. It consists in excising the scarlike tissue layer by layer and piecemeal until soft, less-resistant tissue is encountered. The dissection is carried out on the posterior rather than on the vertebral aspect of the lesion, because collapse toward the mediastinum is desired. The amount of tissue to be excised varies greatly in the different cases. The excision is completed when a well-defined dimple or depression which is characteristic of a sinking in of the wall is felt (and often seen). The cavity has not as yet been entered in any instance, although pulmonary tissue has been cut into on a few occasions. Cavernolysis has been practiced in the majority of the cases, the material consisting largely of extensive and old stiff lesions (average duration of disease five and a half years). Whether or not lysis is employed, operation is terminated by laying the musculoperiosteal flaps in the paravertebral gutter, snugly packing iodoform gauze against the released cavity, drawing the scapula in position, and layer closure of the wound about the gauze pack. The pack is used to steady the mediastinum and to induce a rigid bed at the site of the released cavity. It is usually left in place for six weeks, when it is withdrawn and not replaced. The dead space is found to be occupied by fluid which is slowly absorbed.

The operation can usually be done in one stage for the smaller and softer cavities, but several stages through different approaches may be required for the larger, multilocular, and more resistant lesions. An indirect index of the release of the cavity is the correction of tracheal deviation, partial at least, if it had existed before operation. The only criteria by which I estimate the result of operation are the disappearance of tubercle bacilli from the sputum together with roentgen ray proof of collapse of cavity; the former does not suffice unless supported by the latter. The study of the ordinary film taken to determine the collapse of cavity is amplified by bronchography in order to aid in the interpretation of any areas of rarefaction that may be noted.

The results of operation performed on the lines which have been indicated can be briefly stated. Thirty-four patients have been operated upon (56 operations). Collapse of cavities with bacillus-free

sputum and discharge from the hospital was the result in 16. There was 1 death from contralateral tuberculous pneumonia ten days after operation, probably referable to too widespread a cavernolysis in one stage. One case is closed, with failure to collapse rigid cavities. In another case, an elderly man who is a poor operative risk, the large cavity has been reduced to a narrow cleft but further operation is not advisable. There are 9 cases in which satisfactory progress toward collapse has been made by one or two operations. Of these 1 is probably completely collapsed, 4 are ready for reoperation, and the other 4 are not as yet in condition for further surgery. The remaining 6 patients have been operated upon too recently to make any statement as to interval results. Their clinical course is satisfactory except for 1 case in which postoperative hemorrhagic nephritis has developed. End-results in these patients will be reported at a later time. It is, however, correct to state, I believe, that a case is closed as a surgical problem in pulmonary tuberculosis when collapse of cavities has been achieved. Prolonged survival with uncollapsed cavities cannot be termed any evidence of the efficacy of operation, regardless of the satisfactory condition of the patient.

DISCUSSION

DR. E. J. O'BRIEN (DETROIT).—It is difficult for me to get the logic of this procedure. In the first place, it seems to me it is not based on accurate figures.

The mortality rate, following thoracoplasty, should be much lower than that already encountered in this series of Dr. Neuhof's patients. Also, the series I presented this morning shows a much higher percentage of closure of cavities than is presented here. It seems to me that Dr. Neuhof's work is not consistent with our ideas of the mechanics of collapse therapy or the mechanics of respiration.

You cannot compress a cavity that is being held out under tension, for it runs away from you as the tension is released. In this procedure, as I see it, the removal of the ribs is more extensive than we attempt with thoracoplasty, and then an attempt is made to push down a cavity that tends to recede away from any pressure that is applied to it. I do not believe it possible, except in rare instances, to actually use compression on a cavity until after the lung has been released from tension, and has contracted to normal size. It seems to me that, with this procedure, one is doing a lot of work that is probably unnecessary in the majority of patients, as one cannot tell before operation which patients might not be benefited by the simple removal of the ribs.

DR. HOWARD LILLIENTHAL (NEW YORK CITY).—It seems to me that the outstanding thing that Dr. Neuhof has tried to show us is the collapse of the walls of the cavity itself, not the cavity compressed from without the lung. He goes through the tissues until he reaches a cleavage plane with some danger, of course, of entering the cavity and, although he has not had it happen, of entering the free pleura, not necessarily a fatal accident, but something that we would prefer to avoid.

I can well understand that in certain calcified cases in which one simply cannot succeed from without the lung in achieving a collapse, this method might be particularly valuable. Whether the method should be as universally applied as Dr. Neuhof seems to think, I am not sure, but not having tried it myself, it is hardly

fair for me to criticize it in this way. Even if a cavity of this sort is entered it does not signify failure. It is an extremely difficult thing to keep a tuberculous opening into a cavity open unless it is one which has been opened into an empyema. The cavities which are opened directly from the outside will close in spite of all one can do, and I have one patient whose cavity had to be reopened three different times for drainage. I might be most willing to try this operation in a rigid, calcified case.

DR. ARTHUR M. SHIPLEY (BALTIMORE).—In operating the other day—just about ten days ago—I came upon a cavity high up in the lung, and when I got it exposed after getting away the upper six ribs, the wall of the cavity was quite palpable in the upper lung and it was unyielding. Its walls were fibrotic and very firm.

I wondered if it were permissible to compress that cavity from without. I did not do it; I was afraid to. I just wondered if there was any one in the audience who had attempted such a procedure or if there is any record that such a procedure is permissible. Perhaps Dr. Graham might tell us.

DR. JOHN ALEXANDER (ANN ARBOR).—In the December, 1932, issue of *The American Review of Tuberculosis* Bruns and Casper reported no death in twelve cases of "Casper thoracoplasty," which may be broadly defined as an upper five-rib thoracoplasty with the addition of an extensive extrapleural pneumonolysis with large gauze filling. Colonel Bruns told me recently that his and Casper's last figures show a 32 per cent mortality among 25 patients, seven of the eight deaths having had a direct connection with operation. The chief danger seems to lie in the extensiveness of the one-stage operation and the sudden great collapse of the diseased lung.

My understanding of Dr. Neuhof's presentation is that his operation is somewhat like a Casper thoracoplasty, with the addition of resection of thickened pleurae. His mortality is considerably lower than Casper's but not as low as the modern extensive many-staged extrapleural thoracoplasty, which has demonstrated its ability to close a higher percentage of pulmonary cavities than any other surgical operation of which I have ever heard.

DR. HAROLD NEUHOF (closing).—In connection with Dr. O'Brien's discussion in which he states that he sees no logical reason for what I have proposed, I must assume that the clinical material with which I have come in contact is considerably different from that which he encounters. For example, in the moving pictures he showed us this morning, we saw a very mobile lung field as soon as one or two ribs were excised. That is not the type of field I am accustomed to encounter, because the lesions upon which I operate are, taken by and large, older lesions with more or less stiff-walled cavities surrounded by fibrotic, resistant, and nonmobile pulmonary tissue. In typical instances the only appreciable mobility which I have noted has been mobility lateral to the site of the lesion or lesions.

I am not particularly interested in comparing mortality statistics with Dr. O'Brien. I simply state in passing that in this series of 34 cases there happened to be one death. This is an experience over a period of two years. What may happen to these patients at a subsequent time I do not know. The important point, I think, is, as I have stated, that 16 of these patients were discharged from the hospital with collapsed cavities and with persistently negative sputum. As far as I can see this is the basic purpose in operation for pulmonary tuberculosis.

Dr. Alexander must have misunderstood me when he stated that the method I described consisted in pushing away the lung at the site of cavitation. Dr. Lilienthal has been good enough to correct the impression gained by Dr. Alexander. The primary purpose of my presentation was to indicate my belief that it is necessary

to expose, inspect, or at least palpate the site of the lesion before deciding what procedure should be employed for its treatment. I think that we must all agree that many a case upon which an extensive thoracoplasty has been done has not been appreciably benefited by operation, even when the excision of the thoracic cage has been far more extensive than that described at this meeting or at other meetings. It is certain that one cannot expect complete collapse of cavities when their walls are stiff by merely excising various sections of the thoracic cage. Complete collapse may of course occur but it would have to be classified as an unexpected accident rather than something which could be anticipated. A partial collapse of the cavity is certainly no index of an end-result. So long as appreciable cavitation exists, the problem is essentially what it was before operation.

The procedure which I employ for stiff-walled cavities is not to be confused with the so-called Casper operation which, as I understand it from the description that I read, consisted in a widespread extrapleural release. On the contrary, I believe that the mobilization invited by such extensive extrapleural release is, in a general sort of way, an undesirable and entirely uncalled for procedure. I do not wish to be understood as advocating cavernolysis in every case. That procedure is carried out when a stiff-walled lesion is found and when no appreciable collapse is noted following the customary excision of ribs, no matter how widely such excision is performed.

To answer Dr. Shipley's question, it is in these cases that I have proceeded with efforts at direct release and collapse of the cavities. Pleural adhesions, usually extensive and firm, were encountered at the site of cavitation in all the cases which have come to operation. The fused parietal and visceral pleura, making an appreciably thick mass of tissue, is excised over the site of cavitation until I feel and usually see a depression or sinking in, which indicates that at least the beginning of the collapse of the cavity has been attained. The line of demarcation between scarred pleural and lung tissue is not sharply defined, and I have undoubtedly penetrated into lung tissue superficially upon several occasions. I have noted no untoward results from such superficial penetration of fibrotic lung tissue. The basic principle which I wish to advocate is the exploration of these lesions with an effort at appropriate treatment for them rather than the blind excision of ribs.

INTRAPLEURAL PNEUMOLYSIS

A Critical Review*

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While artificial pneumothorax is the most widely used form of collapse therapy for pulmonary tuberculosis, and while it has greatly reduced the mortality of the disease, it leaves much to be desired as a method of treatment. In too many instances it either cannot be induced, or a successful collapse of the diseased lung cannot be obtained because of pleural adhesions. Numerous statistics have been published which clearly show a direct and constant relation between cures and mortality, and the degree of collapse of the tuberculous lung attained by the pneumothorax.

A partial collapse of a tuberculous lung which does not close cavities nor result in sputum free of tubercle bacilli, offers the patient but little increase in his chances for arrest of his disease. This fact is not appreciated by many phthisiotherapists. Neither do they realize that many of these patients should be given the benefits of other forms of collapse therapy, nor that in some of them the unsuccessful pneumothorax could be converted into a successful one by the operation of intrapleural pneumolysis.

Although twenty years have passed since Jacobaeus[49] first successfully divided intrapleural adhesions by means of a cautery under thoracoscopic control, and many surgeons have accepted and applied this operation, its value as an adjunct to artificial pneumothorax is not universally recognized. I have performed this operation on 15 patients up to April 1, 1933, and I am convinced of its value. I am reporting the results of my cases along with a study of all the cases reported in the literature from 1924 to 1932, inclusive. Such a report of results, with a critical study of complications of the operation and of technical innovations, should prove helpful in establishing the value of this procedure in the treatment of pulmonary tuberculosis.

The first thoracoscope of Jacobaeus was imperfect, but he has since improved it. His present thoracoscope[52] consists of a telescope, size 12 French, with a light on the end, and contains a lens system that gives a right-angled vision. He introduces the thoracoscope through a cannula, previously inserted in the sixth or seventh interspace in the scapular line. He divides the adhesions by means of a galvanocautery introduced through a second cannula inserted between the

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ribs, either in the nipple line or the anterior or midaxillary line. For apical adhesions he inserts the thoracoscope anteriorly and the cautery high in the midaxillary line. At times he also uses a control thoracoscope introduced through a third cannula.

Other men have devised thoracoscopes and accessory instruments and have modified the technic in certain details. Simple direct vision endoscopes have been devised and used by Head[41] and Mascher.[59] Singer[95] inserted a cannula through which he introduced a baby cystoscope and the operating cautery. Thoracoscopes similar in construction to operating cystoscopes and necessitating only one puncture through the chest wall have been devised and are used by Davidson,[18] Chandler,[13] Howe,[114] Kalk,[115] and Kornitzer and Maendl.[53] These instruments give an indirect vision at an angle of either thirty or sixty degrees. Two puncture instruments with an indirect vision at an angle of sixty degrees are used by Pinchin and Morloch,[78] and Cova.[114] Kremer[22] devised a telescope giving a direct vision but he inserts the cautery through a second opening.

Unverricht[101] modified and improved the Jacobaeus thoracoscope and devised a ventilating cannula, and a curved cautery to use in addition. Kremer[22] devised a flexible cannula to admit more easily the curved cautery of Unverricht.

The Unverricht instrument has proved to be by far the most popular one, and really led to the adoption of this operation. It is similar to the Jacobaeus thoracoscope. The size of the telescope is 15 French, and it is equipped with a system of Zeiss lenses giving a right-angled vision. The cannula through which it is introduced is 19 French.

Probably no instrument yet devised is perfect nor has succeeded in making the operation an easy one. Simple direct endoscopes give a too limited field of vision, and cannot possibly permit the operator to reach all the adhesions through one puncture. While they may permit easier orientation, they do not allow the operator to divide all the adhesions near the parietal attachment, which is now known to be of paramount importance.

Any instrument that is inserted through a single opening has three serious defects. First, the cannula must of necessity be large, which diminishes its ease of manipulation and increases the risk of postoperative emphysema. Second, the field of vision is of necessity small in area because the lens system must be reduced in size to make room for the operating instruments in the same telescope. Third, in many cases it will be impossible to reach all adhesions from one opening. The only possible advantage such an instrument can possess is the elimination of the awkwardness of manipulating the cautery through a second opening.

A lens system of direct vision, such as that of Kremer, has the distinct advantages of making orientation much easier, and of permit-

ting the operator to view directly instead of indirectly his operative maneuvers. On the other hand, it requires much more manipulation, and gives a view of only one side of an adhesion, and does not give as extensive a view of the thoracic cavity as does the right-angled telescope. However, Kremer's instrument is a valuable addition to the operator's armamentarium.

The right-angled vision telescope of Jacobaeus and Unverricht requires less manipulation, enables the operator to view almost the whole of the thoracic cavity from one point of introduction, and permits the operator to view more than one side of the adhesion. On the other hand, it makes orientation more difficult, and the manipulation of the operating instruments extremely awkward. Considerable patience and practice are necessary to overcome these difficulties.

In my own experience I have felt the need of a clearer and larger field of vision than the Unverricht thoracoscope gives. Lately I have used the examining telescope of the Stearns-Davis resectoscope. Its size is 18 French, requires a cannula of 23 French for introduction, and it gives a right-angled field of vision of eighty-five degrees as compared to fifty-five degrees of the Unverricht thoracoscope. I believe it is a better instrument, but it is too long, has too long a tip beyond the point of vision, and will have to be modified before it can be adapted to this purpose.

The most important modification that has been made is in the method of dividing adhesions. Kornitzer and Maendl,[53] Davidson,[18] Chandler,[13] and Matson[62] now divide adhesions by electrosurgery. Maurer[65] tried diathermy and did not like it. He was troubled considerably with hemorrhage when using the cautery alone and now he uses a combination of diathermy and cautery. He has devised a combination cautery and electrode and by means of a foot switch can change from the high frequency current to a galvanic current without changing the instrument. He first coagulates a zone around the adhesion in order to control hemorrhage, and then divides the adhesion with the cautery. Jacobaeus[51] states that he has tried the electrosurgical method but has abandoned it for the cautery. So has Unverricht.[104] Matson[62] experienced difficulty in controlling hemorrhage with the cautery and now uses electrosurgery exclusively. He has devised the most useful electrodes that can be obtained for intrathoracic use.

The advantages claimed for electrosurgery are: (1) that the zone of coagulation necrosis is narrower, and that the coagulum is tougher and does not slough off so readily, with less danger of secondary hemorrhage; (2) hemorrhage is less likely to occur and if it does, it can be more readily and accurately controlled; (3) the smoke and

heat from the cautery are eliminated, thereby reducing the incidence of postoperative effusions; and (4) the operation is speeded up by electrosurgery.

I use the electrosurgical method and believe it is superior to the cautery method. However, it has two disadvantages. In the first place, division of an adhesion is more painful when done by electrosurgery, and second, a very expensive apparatus is required. A small and less expensive machine will not be found satisfactory for dividing adhesions. I have no doubt that the failure of some men to obtain satisfactory results was due to poor equipment and not to the method. I have found the Davis-Bovie unit to be perfectly satisfactory for the division of adhesions. I use the electrodes designed by Matson.

In addition to the modifications of instrument and method, certain accessory instruments have been devised that may prove very useful at times. Rapp,[114] Heine,[42] Maurer[65] and Bethune[5] have devised illuminating sounds for the purpose of transilluminating adhesions in order to ascertain more readily the presence of lung tissue or the prolongation of cavities within them. Chandler,[13] Pinchin and Morloch,[78] and Maurer[65] have devised needles with which to anesthetize adhesions under thoracoscopic control. I have used the Denman needle for splanchnic anesthesia very satisfactorily for the same purpose. Jacobaeus[52] has devised a hooked cautery, which he says facilitates the division of small string-like adhesions. All of these instruments, while not necessary, should prove very helpful to the surgeon.

Considerable difference of opinion exists as to the point of election for insertion of the thoracoscope and the cautery or electrode. Jacobaeus,[49] Unverricht,[101] and Matson[60] state that in the majority of cases they introduce the thoracoscope posteriorly and the operating instruments anteriorly, for here the interspaces are wider and permit easier manipulation. Unverricht[101] introduces the cautery low in the anterior or midaxillary line. Diehl and Kremer[22] state that the thoracoscope should be introduced anteriorly and the cautery in the posterior axillary line. Maurer[64] states that the thoracoscope should always be introduced so that the optical system is parallel to the main adhesion, which more frequently will be in the mammary line or anterior axillary line. He further states that the cautery should be introduced so that it is vertical to the main adhesion, which will, as a rule, be in the axillary line. For apical adhesions all agree that the thoracoscope should be introduced in the second interspace anteriorly, and the cautery high in the axilla. There can be no set rule as to the point of introduction of the thoracoscope. One has to be guided by a careful study of stereoroentgenograms as to where the thoracoscope shall be introduced, and decide the point of introduction of the electrode or cautery after his examination with the thoracoscope. Not infrequently I find that it helps to interchange the instruments in

order to reach all of the adhesions, and in order to do this readily, one should have interchangeable cannulas, which the Unverricht apparatus does not have. (The later model of the Unverricht thoracoscope is equipped with interchangeable cannulas.)

The indication for the operation is the presence of an incomplete pneumothorax of from three to six months' duration, which, on account of string, cord, or band-like adhesions has not produced a satisfactory collapse of the diseased lung. Cavities which are not closed, positive sputum, and in some cases the occurrence of distressing pain and cough after refills, constitute an unsatisfactory collapse.

Contraindications are: (1) the presence of an acute febrile effusion, either serous or purulent, and (2) the presence of active extensive disease in the other lung, in which case there is no reasonable hope of cure even with a complete collapse on one side. Chronic, afebrile, serous, or purulent effusions are not contraindications according to Matson,[60] but they do increase the risk of pleurocutaneous fistula and of injury to the lung. All operators of experience definitely state that pneumothorax should be given a trial of at least from three to six months, because at times adhesions may stretch sufficiently for cavities to close and because the pleura gradually becomes less irritable after a longer period of pneumothorax and less likely to react severely to operative trauma.

X-ray studies are of great help in choosing cases for operation, but they are often misleading as to the number and character of adhesions. Their operability can only be determined by thoracoscopic examination. Unfortunately, all of these cannot safely be divided. Only string, cord, or band-like adhesions can be cauterized with safety. Short, thick, broad adhesions and surface synechia, as a rule, contain pulmonary tissue and are dangerous to divide.

The percentage of partial collapse cases that are suitable for pneumolysis is not known. Matson[62] claims that in his experience 45 per cent of his cases of partial pneumothorax are suitable for pneumolysis. On the other hand, O'Brien[75] states that the need for intrapleural pneumolysis seldom arises. In sanatoriums with which he is connected, over two thousand pneumothorax refills are given every month, yet in five years' experience he has found it necessary to perform pneumolysis on only 16 patients. He prefers to supplement pneumothorax with phrenic neurectomy, and wait months in which to effect closure of cavities. This opinion of O'Brien and his associates is hard for me to understand, because probably no other group in America uses collapse therapy more extensively than they. Over 70 per cent of all their patients receive some form of collapse therapy early in the course of their treatment. O'Brien does not believe in giving patients prolonged conservative treatment before instituting pneumothorax, and yet he does believe in giving a partially success-

ful pneumothorax months to effect closure of cavities. Kremer[54] states that intrapleural pneumolysis was done in 8.6 per cent of his cases of artificial pneumothorax, and Pomplum[80] performed the operation in 14 per cent of his cases. I believe that the experiences of Kremer and Pomplum are nearer the average than that of either Matson or O'Brien.

Results of the operation are judged by the success of the procedure in converting a clinically unsuccessful pneumothorax into a successful one. The end-results of the operation belong to that of artificial pneumothorax, and yet in the final analysis the end-results should be the real test of the operation.

I have collected from the literature 1,835 cases in which results were reported, and to this number I have added 15 cases of my own. The results are tabulated in Table I. Technical success is not always followed by clinical success, or technical failure by clinical failure. Occasionally, a stiff-walled cavity will not close, and not infrequently the division of a few adhesions or of a broad band will be followed by a clinical success. Briefly stated, the immediate results of the operation in the 1,850 reported cases are as follows:

Clinically successful	75.5%
Clinically unsuccessful	24.5%
Patients made worse by operation	4.5%
Patients died from operation	1.08%

Schelenz[90] states that at Sommerfeld the good results from artificial pneumothorax have risen from 48.5 per cent in 1921, to 72.5 per cent in 1927, by the use of intrapleural pneumolysis.

Only Unverricht[103] has reported late results from two to nine years after pneumothorax ended. Of 247 cases of full technical success 151, or 61 per cent, were cured and able to work. Of 49 cases of incomplete success 16, or 32 per cent, were cured and able to work. These results are a great improvement over the results reported by Matson[60] for cases of partial collapse without the aid of pneumolysis. Of his cases of incomplete pneumothorax, only 13 per cent were well and 13 per cent arrested from one to ten years after treatment was begun.

Unquestionably, this study of the results of operators of varying experience and skill, definitely establishes the operation of closed intrapleural pneumolysis as being the most valuable adjunct to artificial pneumothorax that we have. Not only will it enhance the brilliant results of artificial pneumothorax, but it will save many patients from the more drastic operation of thoracoplasty.

A study of the complications of the operations will prove helpful to all surgeons who are doing this work. As the operation is not without risks, the surgeon should thoroughly understand what they are and how to avoid them.

TABLE I

RESULTS OF CLOSED INTRAPLEURAL PNEUMOLYSIS

AUTHOR	METHOD	NUMBER OF CASES	COLLAPSE TECHNICALLY SUCCESSFUL	COLLAPSE TECHNICALLY UNSUCCESSFUL	COLLAPSE CLINICALLY SUCCESSFUL	COLLAPSE CLINICALLY UNSUCCESSFUL	PATIENTS MADE WORSE BY OPERATION	PATIENTS DIED FROM OPERATION
Arce[2]	Cautery	1	1	0	1	0	0	0
Barnes[4]	Cautery	2	2	0	2	0	0	0
Bigger[7]	Cautery	18	18	0	14	4	1	0
Brissaud[9]	Cautery	50	47	3	25	25	8	4
Couland[17]	Cautery	39	21	18	26	13	2	0
Dahlstedt[113]	Cautery	1	1	0	0	1	0	1
de Carvalho[19]	Cautery	3	3	0	3	0	0	0
de Castiglione[20]	Cautery	25	14	11	19	6	4	0
de Paula[21]	Cautery	2	2	0	2	0	0	0
Diehl and Kremer[22]	Cautery	272	171	101	193	79	13	2
Duoady and Contiades[26]	Cautery, now combined	8	5	3	4	4	1	0
Fisher[30]	Cautery	16	9?	7?	9	7	0	0
Forsee[31]	Cautery, now electrosurg.	26	21	5	16	10	5	3
Gehrke[113]	Cautery	4	3	1	3	1	0	0
Gravesen[113]	Cautery	16	9	7	7	9	2	2
Gullbring[34]	Cautery	40	24?	16?	24	16	2	0
Haberlin[38]	Cautery	6	5	1	5	1	0	1
Head[40]	Cautery	4	2	2	2	2	0	0
Heymer[44]	Cautery	200	104	96	179	21	?	0
Holmboe[113]	Cautery	12	7	5	7	5	1	1
Jacob and Couland[47]	Cautery	1	1	0	1	0	0	0
Jacobaeus[49]	Cautery	150	121	29	99	51	13	3

TABLE I—CONT'D

AUTHOR	METHOD	NUMBER OF CASES	COLLAPSE TECHNICALLY SUCCESSFUL	COLLAPSE TECHNICALLY UNSUCCESSFUL	COLLAPSE CLINICALLY SUCCESSFUL	COLLAPSE CLINICALLY UNSUCCESSFUL	PATIENTS MADE WORSE BY OPERATION	PATIENTS DIED FROM OPERATION
Korbsch[113]	Cautery	3	3	0	3	0	0	0
Kornitzer and Waltuch[53]	Combined	50	43?	7?	43	7	2	0
Leendertz[57]	Cautery	12	4	8	8	4	0	0
Matson[63]	Cautery, now electrosurg.	206	124	82	138	68	4	1
Merlo[69]	Cautery	5	4	1	5	0	0	0
Peters and Cornish[77]	Cautery	36	27	9	33	3	1	0
Pomplum[80]	Cautery	65	49	16	61	4	?	0
Rosal and Caralps Masso[85]	Combined	6	6	0	6	0	0	0
Sachs[86]	Cautery	2	2	0	2	0	0	0
Sarajon[88]	Cautery	1	1	0	1	0	0	0
Schröder[91]	Cautery	7	7	0	6	1	0	0
Somne[113]	Cautery	11	9	2	7	4	?	0
Tegtmeier[98]	Cautery	27	20?	7?	20	7	2	0
Unverricht[103]	Cautery	334	264?	70?	264	70	14	0
Voute[107]	Combined	18	14	4	13	5	0	0
Weber[109]	Cautery	5	5	0	5	0	0	0
Welles[110]	Cautery	141	122?	19?	122	19	8	0
Xalabarder[111]	Cautery	2	2	0	2	0	0	0
Zoboli[112]	Cautery	8	8	0	8	0	0	0
Total from literature		1,835	1,305	530	1,388	447	83	18
Author's cases	Electrosurg.	15	10	5	9	6	1	1
Total all cases		1,850	1,315	535	1,397	453	84	19
Percentage			71%	29%	75.5%	24.5%	4.5%	1.08%

TABLE II

COMPLICATIONS OF CLOSED INTRAPLEURAL PNEUMOLYSIS (1924-1933)

AUTHOR	METHOD	NUMBER OF CASES	HEMORRHAGE		SMALL EXUDATES	LARGE SEROUS EXUDATES OFTEN TBC.	TUBERCULOUS EMPYEMA	MIXED INFECTED EMPYEMA	PLEUROCUTANEOUS FISTULA	REEXPANDED LUNG	EMBOLISM	SECONDARY PERFORATION	OTHER COMPLICATIONS
			SMALL	SERIOUS									
Arce[2]	Cautery	1	0	0	0	0	0	0	0	0	0	0	0
Barnes[4]	Cautery	2	0	0	0	0	0	0	0	0	0	0	0
Bigger[7]	Cautery	18	1	0	?	0	0	1	0	0	0	0	0
Brissaud[9]	Cautery	50	0	0	?	2	4	4	0	2	0	2	0
Couland[17]	Cautery	39	4	1	27	0	0	0	0	1	0	1	0
Dahlstedt[113]	Cautery	1	1 fatal	0	0	0	0	0	0	0	0	0	0
de Carvalho[19]	Cautery	3	0	1	0	0	0	0	0	0	0	0	0
de Castiglione[20]	Cautery	25	0	1	10	3	2	2	0	0	0	0	Mediastinal emphysema—1
de Paula[21]	Cautery	2	0	0	0	0	0	0	0	0	0	0	0
Diehl and Kremer[22]	Cautery	272	8	5	15	18	8	3	0	0	1	2	0
Duoady and Contiades[26]	Cautery Combined	8	1	1	0	2	0	1	0	0	0	0	0
Fisher[30]	Cautery	16	0	0	5	0	0	0	0	0	0	0	0
Forsee[31]	Cautery Electrosurg.	26	1	0	14	0	2	6	2	1	0	0	0
Gravesen[113]	Cautery	16	0	0	0	0	0	2	0	0	0	0	0
Gullbring[34]	Cautery	40	0	0	?	?	0	1	1	0	0	0	0
Haberlin[38]	Cautery	6	0	0	0	0	0	1	0	0	0	0	0
Head[40]	Cautery	4	0	0	0	0	0	0	0	0	0	0	0
Holmboe[113]	Cautery	12	0	0	?	?	0	1	0	0	0	0	0
Jacob and Couland[47]	Cautery	1	0	1	0	0	0	0	0	0	0	0	0
Jacobaeus[49]	Cautery	150	0	1	0	21	11	3	0	2	0	0	0

TABLE II—CONT'D

AUTHOR	METHOD	NUMBER OF CASES	HEMORRHAGE		SMALL EXUDATES	LARGE SEROUS EXUDATES OFTEN TBC.	TUBERCULOUS EMPYEMA	MIXED INFECTED EMPYEMA	PLEUROCUTANEOUS FISTULA	REEXPANDED LUNG	EMBOLISM	SECONDARY PERFORATION	OTHER COMPLICATIONS
			SMALL	SERIOUS									
Kornitzer and Waltuch[53]	Combined	50	0	0	?	?	0	2	0	0	0	0	0
Leendertz[57]	Cautery	12	0	0	?	?	0	0	0	0	0	0	0
Matson[63]	Cautery Electrosurg.	206	0	3	144	19	0	4	0	0	0	0	0
Maurer[68]	Combined	160	?	?	44	45	1	0	0	0	0	1	0
Merlo[69]	Cautery	5	0	0	0	0	0	0	0	0	0	0	0
Peters and Cornish[77]	Cautery	36	0	0	?	?	0	1	0	0	0	0	0
Rosal and Caralps Masso[85]	Combined	6	0	0	1	2	0	0	0	0	0	0	0
Sachs[86]	Cautery	2	0	0	0	0	0	0	0	0	0	0	0
Sarajon[88]	Cautery	1	0	0	0	0	0	0	0	0	0	0	0
Schröder[91]	Cautery	7	0	0	0	0	0	0	0	0	0	0	Brachial neuritis—1
Tegtmeier[98]	Cautery	27	0	0	25	4	2	0	0	0	0	0	0
Unverricht[104]	Cautery	650	0	0	114	39	9	2	0	0	0	2	0
Voute[107]	Combined	18	0	0	?	?	0	0	0	0	0	0	0
Weber[109]	Cautery	5	0	0	0	0	0	0	0	0	0	0	0
Welles[110]	Cautery	141	0	0	55	9	6	2	0	0	0	0	0
Xalabarder[111]	Cautery	2	0	0	0	0	0	0	0	0	0	0	0
Zoboli[112]	Cautery	8	1	0	1	0	0	0	0	0	0	0	0
Total from literature		2,028	16	15	455	164	45	36	3	6	1	8	
Author's cases	Electrosurg.	15	0	0	5	1	1	1	0	0	0	0	Pleural shock—1
Total all cases		2,043	16	15	460	165	46	37	3	6	1	8	
Percentage			0.77%	0.73%	22.5%	8.07%	2.2%	1.8%	0.14%	0.29%	0.04%	0.39%	

In Table II are listed 2,043 cases, including my own, in which the complications were given. The reason for the difference in the number of cases in the two tables is that Unverricht[104] has reported the complications in 650 cases and results in only 334 cases, Maurer[68] reported the complications and not the results in 160 cases, and several others failed to mention complications. It will be of interest to discuss the complications in detail.

Hemorrhage.—In many instances a slight ooze, either from the adhesion itself or from the puncture wound, occurs, but it is of no consequence. Sixteen cases of small hemorrhage and 15 cases of serious hemorrhage were reported. Of these, one (that of Dahlstedt[116]) was fatal. Jacob and Couland[47] reported a life threatening hemorrhage from an intercostal artery injured by the trocar. Nearly all of the reported hemorrhages occurred with the use of the cautery. The percentage of hemorrhage is 1.5 per cent, with only one fatal hemorrhage having been reported. Unverricht,[104] Welles,[110] Diehl and Kremer,[22] and Jacobaeus[49] have learned to prevent hemorrhage with the cautery by using a very low heat. They emphasize that the cautery should never be hotter than a dull red. Matson[62] had three serious hemorrhages with the cautery method and has abandoned the cautery for electrosurgery, which he states makes hemorrhage less likely to occur, and more easily controlled when it does occur. Maurer[65] experienced difficulty in controlling hemorrhage with the cautery. He then used diathermy, which he found unsatisfactory in dividing adhesions. He now uses a combination of the two methods. He first coagulates a narrow zone with diathermy and then divides the adhesion through the coagulated zone with the galvanocautery. Bethune[5] suggested and used silver clips on adhesions to prevent hemorrhage. It seems to me that Bethune's suggestion would be far more valuable in preventing opening of cavities than hemorrhage, for it is easy to control hemorrhage with electrosurgery, but electrosurgery offers no safeguard against injury to the lung.

Exudates.—Small serous exudates occurred in 460, or 22.5 per cent of all cases, and are of little consequence. Large and more persistent serous exudates, many of which were tuberculous effusions, occurred in 165, or 8.07 per cent of all cases. Exudates are said by some to be the result of smoke and heat from the cautery, while others believe they result from blood in the pleural cavity. Tuberculous effusions are the result of opening tuberculous foci in adhesions. These cannot be avoided, although Matson[62] claims that electrosurgery diminishes this risk. The tuberculous effusion and tuberculous empyema of my series were in cases done with the electrosurgical methods.

Tuberculous Empyema.—Some of the serous tuberculous effusions progressed into tuberculous empyemas in 46, or 2.2 per cent of the reported cases. The one case of mine had a preexisting afebrile serous

effusion. After two operations at which a total of nineteen adhesions were divided, the effusion gradually became purulent. After six months it subsided. According to Head,[40] tuberculous effusions are only twice as frequent following pneumolysis as they are in the course of artificial pneumothorax. Tuberculous empyema may defeat the good result of the operation and may call for further surgical measures to control it.

Mixed Infected Empyema.—The most serious of all complications is a mixed pyogenic and tuberculous empyema that occurred in 37, or 1.8 per cent of all cases. All reported have been due to perforation of the lung or the opening of cavities. The majority of deaths were due to this complication. Even if it does not prove fatal, empyema leaves the patient in a much worse condition than before operation. Gloyne,[33] Franz,[32] Maurer,[64] and others have demonstrated that two-thirds of all adhesions may contain tuberculous foci, caseated areas, degenerated pulmonary tissue, or prolongations of cavities. It is not always possible for the operator to determine with accuracy whether the adhesion contains caseated areas or pulmonary tissue, or not. Jacobaeus[49] states that, in his opinion, pulmonary tissue is frequently cauterized but that no infection results because of the retraction and contraction of the stump of the adhesion and of the lung.

Maurer[64] has classified adhesions into four types as follows:

1. Adhesions of which neither the pulmonary nor the thoracic insertion is broadened out. These consist mainly of strings and fine cords, do not contain pulmonary tissue, but are not of great technical importance.

2. Adhesions of which the thoracic end is broadened. These do not contain pulmonary tissue, and are not common.

3. Adhesions of which the pulmonary insertion is conical. These always contain pulmonary tissue or prolongations of cavities for a varying distance. They are quite common, are more dangerous to section, and are of great technical importance.

4. Adhesions of which both the thoracic and pulmonary insertions are expanded like an hourglass. There is a more or less distinct line of demarcation between the pulmonary and thoracic portions, beyond which no pulmonary tissue is present. They are not uncommon and are of great technical importance.

Maurer[64] further claims that inasmuch as the operator cannot with accuracy tell where lung tissue ends in the adhesions, they should never be divided, but should always be enucleated at the parietal insertion into the thoracic wall. Maurer first coagulates a zone around the adhesion at its thoracic insertion with diathermy, and then divides, or rather strips, it from the chest wall with the cautery. He has reported 160 cases without a single case of empyema as a result of injury to the lung. His one case of empyema was the result of a sec-

ondary perforation of the lung and not the result of the operation. He is the only operator of large experience who has avoided this complication. His method is technically more difficult to do; it increases the danger of hemorrhage, and it is more painful and requires anesthesia of the adhesion itself. Yet, it definitely increases the usefulness of the operation and greatly reduces the danger of empyema. I believe Maurer has made a most important contribution to the technic of the operation.

The average operator can only avoid this dangerous complication by careful orientation and conservatism. One of my earlier experiences resulted in empyema from perforation of the lung, the result of an ill-advised attempt to divide a crescentic band too deeply. Until one can master the technic of the operation, conservatism should be his creed.

Pleurocutaneous fistula has occurred in 3 cases, or 0.14 per cent. It occurred in cases in which there was a preexisting effusion. In such cases the effusion should be frequently aspirated, and the patient should be kept lying on his sound side until the puncture wounds have healed.

Reexpansion of the lung with loss of the pneumothorax has occurred in 6 cases, or 0.29 per cent. The majority of the patients have a slight subcutaneous emphysema. A pressure bandage over the wounds and control of cough by opiates will, in a measure, control emphysema. Occasionally, however, the loss of air will be rapid and emphysema extreme. Recently, I had this to occur in one of my patients not recorded in this report. I had divided ten adhesions, but I was not able to control a hacking cough, even with large doses of morphine. The danger of loss of pneumothorax was recognized, and frequent refills were given. They were kept up until his subcutaneous emphysema became so extreme that his physician, Dr. Ringer, and I felt that it was wiser to abandon the pneumothorax than to keep increasing his emphysema. This experience was discouraging to say the least.

Secondary perforation of the lung, weeks after the operation, has been reported in 8 cases, or 0.39 per cent. It is probably in no wise more frequent than during the course of an artificial pneumothorax. Deaths resulting from these secondary perforations have not been included in the mortality statistics in this series. However, I have not considered perforations as secondary unless they occurred later than six weeks after operation, or were proved by autopsy not to have been caused by the operation.

Pleural shock has occasionally been mentioned as occurring, but no fatal cases were reported. *Mediastinal emphysema* was reported by de Castiglione,[20] and one case of *brachial neuritis* was reported by Sarajon.[88]

One fatal case of *cerebral embolism* was reported by Diehl and

Kremer,[22] and de Castiglione[20] stated that 3 fatal cases were reported by Leotta, Saugmaun and Skäergord. In order to lessen the danger of gaseous embolism, Matson[61] advises that the first reinflation be made with carbon dioxide.

Two complications occurred in my series which are worth mentioning. One patient, who had extensive bilateral disease and on whom only a thoracoscopic examination was done, developed an acute pulmonary edema thirty-six hours after operation. No pressure changes had occurred in the pneumothorax to account for the failure of his right heart. The patient recovered, but died several weeks later from his tuberculosis. In another patient, who was in excellent general health, I divided five adhesions successfully. At the conclusion of the operation I inserted into the pleural cavity 10 c.c. of a 2 per cent mercurochrome solution. He immediately complained of severe pain in his chest, became pale, and his pulse became very weak. These symptoms subsided in five minutes. Evidently, I produced a pleural shock by injecting a mercurochrome solution that was not warmed. At the close of each operation I now inject 10 c.c. of a warmed hexylresorcinol solution very slowly, and have had no trouble from it. Inasmuch as one's technic cannot be absolutely perfect with endoscopic instruments, I believe the insertion of some antiseptic solution adds a safeguard against infection.

In spite of the good results reported and the relative safety of the operation of closed intrapleural pneumolysis, there are a few surgeons who condemn the operation as being not only technically difficult, but extremely dangerous. They prefer the open method of intrapleural pneumolysis. Rosving and Saugmaun are credited by de Carvalho[19] as being the first to attempt open pneumolysis in 1913, and since then sporadic reports of this operation have been made. I have been able to find a total of 52 cases reported in the literature available. I know this list is not complete, as it does not contain cases said to have been reported by Leotta, Paternoster, and Sanza, whose articles were not available. The largest series of the open operation is the 16 cases reported by O'Brien to this Society last year.

Table III lists the 52 cases. Of them, 30, or 57.6 per cent, were clinically successful. Severe hemorrhage occurred in 3 cases, or 5.7 per cent. Empyema developed in 15 cases, or 28.8 per cent. Ten, or 19.2 per cent, of the patients were made worse by the operation, while 10, or 19.2 per cent, died directly as a result of the operation.

Eloesser[28] uses a trapdoor incision. Carter[12] resects from four to six inches of a rib and uses a rib spreader to enlarge the opening. O'Brien[75] resects a short piece of rib and makes an incision slightly more than an inch in length through the rib bed. All but Carter[12] divide the adhesions between clamps and ligate if necessary. Carter enucleates the parietal insertion of the adhesion from the chest wall and sews over

the stump of the adhesion to cover raw edges. The parietal stump is left bare. He does the same thing in the open operation that Maurer[64] does in the closed operation.

O'Brien[75] claims that the advantages of this operation are: (1) that the thoracic cavity can be readily explored by direct vision, and adhesions are made more accessible; (2) that there is less danger of infecting the pleural cavity; (3) that it is less time consuming; and (4) that there is less danger of hemorrhage.

The technic of the open operation is not standardized, and no one has performed the operation in a large enough series of cases to have established the technic, nor the morbidity and mortality rates, as well as the end-results of the open operation. For that reason it is not quite fair to compare the two methods. However, I see no other way to make a

TABLE III

RESULTS OF OPEN INTRAPLEURAL PNEUMOLYSIS (1924-1933)

AUTHOR	NUMBER OF CASES	SUCCESSFUL	SEVERE HEMORRHAGE	EFFUSION	EMPYEMA	MADE WORSE BY OPERATION	DIED FROM OPERATION	EXTENSIVE EMPHYSEMA
Collected by John Alexander[1]	17	5	3	?	4	5	3	?
Bettman[6]	1	1	0	1	0	0	0	0
Carter[12]	4	4	0	?	0	0	0	1
Eloesser[29]	4	3	0	1	1	1	0	?
O'Brien[75]	16	12	0	?	3	1	3	2
Sachs[86]	2	0	0	0	2	2	0	0
Sauerbruch[89]	6	2	0	0	4	1	3	0
Watson[108]	2	1	0	0	1	0	1	0
Total	52	30	3	?	15	10	10	3
Percentage		57.6%	5.7%	?	28.8%	19.2%	19.2%	5.7%

comparison, except to stick to the recorded figures, and contrast the results of the fifty-two open operations reported by several surgeons with the results of nearly two thousand cases of the closed method reported by many surgeons, some of large experience, and many with very little or no experience.

A comparison of the reported results of the two operations disprove each of these advantages. The open operation is more shocking and more likely to be followed by extensive emphysema; empyema occurred in 28.8 per cent of the open operations in contrast to 1.8 per cent in the closed operation; and hemorrhage occurred in 5.7 per cent of the open method as against 1.5 per cent in the closed method, the mortality of the open pneumolysis was 19.2 per cent, while it was only 1.08 per cent in the cases of closed pneumolysis.

While my own experience has been limited to the closed method, I am convinced by a study of the experience of others that the open operation of intrapleural pneumolysis is more dangerous than thoracoplasty. It should be reserved for those patients in whom it is not safe

to divide the adhesions by aid of the thoracoscope, and on whom, because of other considerations, thoracoplasty cannot be performed. The method of Carter[12] appeals to me as being the safest technic of the open operation to follow.

In conclusion, it is my opinion that the thoracoscope of Unverricht is the best instrument for the operation of closed intrapleural pneumolysis, and that the electrosurgical method is superior to the cautery method. I also believe that Maurer's technic in dividing an adhesion at its parietal insertion should extend the field of indications of the operation and greatly reduce the incidence of empyema.

A study of the reported results of the closed method definitely establishes not only its value as an adjunct to artificial pneumothorax therapy of pulmonary tuberculosis, but also its greater safety and effectiveness over the open operation.

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DISCUSSION

DR. DRASH.—I would simply like to add to Dr. Moore's paper, our experience with this method in the state of Virginia. We have been using the Davidson thoracoscope in conjunction with the Bovie electrosurgical unit. These cases have been obtained from the Blue Ridge and Catawba Sanatoriums, which are institutions for the care of tuberculous white patients in Virginia.

There were 34 cases in which actual operation was done. In 4 other cases the instrument was inserted into the thoracic cavity, but no attempt was made to divide adhesions. This was either because of extensive pleural tuberculosis or presence of so many or such large adhesions which were not visible in the x-ray, making operation impossible. Seven of these cases were done too recently to include in the summary, so we are basing our figures on 27 cases. The earliest case was done in October, 1931; these 27 cases brought the figures up to February 1, 1933. Of that number in which we were able to cut adhesions that were directly over the tuberculous area, those cases were considered as technically successful. In most instances we were able to divide all of the adhesions in the pneumothorax cavity. In some instances they were not all cut and this was not considered necessary when we were able to divide those overlying tuberculous areas, permitting the necessary collapse.

This is an analysis of clinical results based on fever, cough, sputum, weight, and efficiency of pneumothorax. Twenty-four of these 27 patients were definitely improved, 88 per cent; 3 were unchanged, 11 per cent.

Analysis of weight; 14 patients gained weight; 10 patients remained stationary; 3 lost weight. These 3 patients were well overweight and lost five pounds each; yet their clinical condition was definitely improved.

Nineteen patients had positive sputum before operation, 9 patients after; that is, the sputum in 10 cases changed from positive to negative (52.5 per cent).

Complications: One hemorrhage occurred. This was certainly considerably less than 100 c.c. The bleeding was stopped by coagulation. This patient was fluoroscoped soon after operation, within two days. He had had a little fluid previously; there was no perceptible difference in the fluid level.

One patient developed sufficient fluid after operation to require one aspiration. There were no cases of pyogenic or tuberculous infections in this series, and no mortality. We are including in that also the 7 cases which we did not include in the other figures; there are no other complications so far.

Our conclusions are that the procedure is quite safe, subject, of course, to the limitations Dr. Moore has mentioned. We have been able to divide 85 per cent of the adhesions, and nearly 90 per cent of the patients operated upon were definitely improved.

DR. E. J. O'BRIEN (DETROIT).—It is quite a novelty to me to be accused of being conservative. I think, however, that Dr. Moore is a bit confused about my viewpoint on pneumolysis.

I do not think it is quite true that I believe in leaving cavities open needlessly over a period of months. I believe they should be closed as soon and as safely as possible, but I do not believe in meddlesome interference with adhesions, until I am sure they are preventing this closure.

Before writing the paper to which Dr. Moore refers, I checked over 500 patients who were under pneumothorax treatment to see whether or not we had been too conservative in our treatment of patients with open cavities and adhesions which were

apparently preventing proper collapse early in the course of treatment. We found only three in which we thought it would have been better if we had severed adhesions earlier; one of those had already been voted on for this procedure.

It is my contention that a great many adhesions that are cut are not holding open cavities. The detection of the presence of open cavities and adhesions by the roentgenogram does not mean at all that the adhesions are holding open the cavity. A thick-walled cavity often resists closure until complete collapse of the lung is obtained. Cavities in consolidated lungs are also difficult of closure. The presence of adhesions in these conditions may have no bearing on the collapse. I have seen numbers of pictures following the severance of these adhesions in which there was no change in the cavity, and only after months of pneumothorax treatment was effective closure obtained. In my opinion, the severance of adhesions in these patients accomplished nothing. Many adhesions are easily stretched under negative pressures, and others with the use of positive pressures. I believe an attempt to overcome adhesions by the addition of phrenic nerve operations and increased pressures should always be made before resorting to pneumolysis.

I believe, also, that pneumothorax treatment should be carried on for at least six months before any attempt is made to cut adhesions, because I do not believe that one can tell before that time whether or not a cavity is going to be closed without this procedure. If a cavity remains stationary during the course of pneumothorax treatment, and adhesions are preventing proper closure, I believe they should be cut.

The dangers in stretching adhesions, I find, are not very great. At the meeting of the American Therapeutic Association next month, Dr. Benson, my associate, and I are reporting a series of 99 patients in whom spontaneous pneumothorax occurred at the Herman Kiefer Hospital. In none of these did the accident occur following the attempt to stretch adhesions. I think this justifies the belief that the gradual stretching of adhesions, which can be accomplished usually under negative pressures, is not dangerous.

In Dr. Moore's summary of the results of open pneumolysis, he made no attempt to specify the type of operation employed; "open pneumolysis" may mean anything from the small incision used by me to the extensive operation advocated by Carter. I can see no particular difference between inserting two trocars, or making a small opening into the pleural cavity, which can easily be sutured.

The expression, "closed pneumolysis," is a misnomer. It is just as much an open pneumolysis as is my method. The opening that I make, in the majority of cases, is not larger than the combined opening of the two trocars used in the so-called "closed method." With my operation, you can see the adhesion directly, put on your champs, crush it, use electrocoagulation, and then sever the adhesion with cautery knife. If bleeding occurs, you can control it.

Dr. Moore stated that three empyemas resulted from the open operation in my series of patients. This is not exactly correct. One of these occurred months later under pneumothorax treatment, and another occurred after the wound was opened, during a coughing spell, in a patient whose pressures were made highly positive by mistake two days after the operation. I do not believe that the empyema in either of these two patients can be attributed to the operation itself.

So the results, I think, from the pneumolysis as it is performed through this very small incision will be just as favorable as those reported here. I, however, am less prejudiced against the blind cutting of adhesions since electrosurgical methods are used. It seems to me that it is of more importance to know when an adhesion should be cut. It is of no interest to me that hundreds of adhesions have been cut with no mortality if it was unnecessary to cut them.

DR. LEO ELOESSER (SAN FRANCISCO).—I have not done one of these extensive open operations that were first proposed by Dr. Torek for many years, although I may say that of five patients upon whom I did operate, some of whom had a stormy course, all recovered perfectly and all are still alive. If I am not mistaken, the oldest one dates from ten years ago, so the procedure may not be so bad as it would seem on the face of it.

I think that Dr. O'Brien is quite correct when he says that merely seeing an adhesion in the x-ray does not mean that that adhesion is keeping a cavity open. Some of them do and some of them do not. Occasionally, you will burn one through and you will see the strand pop and the cavity will fly away from the chest wall; but often they are quite loose and when you touch them they flap around like a sail in the wind, so to speak.

The apparatus that I have seen hitherto has not entirely satisfied me. I started in with a Mandl-Kornitzer apparatus, which is good as far as its optical system is concerned, but poor, I think, in its operating part. As Dr. Moore said, there is a part in it which carries a high frequency current, and another part that carries an electrocautery current. This instrument was made by a urologist. The parts are so tiny that I will warn you against using them, because I was unfortunate enough the first time I connected the diathermy to a good American machine to burn the electrode off, so that one of my patients still has the electrode in her chest. Therefore, I had another one constructed that is about halfway between Matson's and the Mandl-Kornitzer apparatus. It has the Mandl-Kornitzer optical part, which gives a 60° angle. I think that that angle is more efficient than the right-angled one. It gives a much wider field and it allows one to work more directly.

On the other hand, I had a bakelite cannula constructed something like Matson uses, so as to insulate the electrode where it enters the chest. I had an electrode constructed by the Westinghouse people, which is very satisfactory indeed. It is something like the Viennese electrode, but much heavier. It has a good, strong watch spring. The spring is fitted to a strong bakelite handle. One can curl the watch spring around an adhesion and then turn on the coagulating current and watch the adhesion coagulate under the eye. When the coagulation is completed, one can turn on the cutting current and burn the adhesion through. It is such a delightful plaything that it is almost impossible to resist the temptation to use it more often than one should.

DR. NORMAN BETHUNE (MONTREAL).—This is a very beautiful, critical review of the subject. It is a tremendously fascinating subject, especially to those attracted by delicate surgical technic. It looks so simple. It may not be simple; it may be dangerous. I do not think it should be in everybody's hands.

I think there is a point that has not been raised this morning: namely, that because adhesions are holding out a cavity, those adhesions should be cut and the lung collapsed. There are some adhesions holding out cavities which should not be cut, even though it is a temptation to cut them. I believe that a thoracoplasty, which is a permanent and satisfactory operation, should be done in some cases instead of inviting the risk of pneumolysis.

In 50 cases of my own I have had 3 cases of empyema; 2 of those patients required drainage. They will eventually require thoracoplasty. I have subjected them to this complication with the consequent drain on their resistance, and regretted it. I think there are certain large cavities about which one may say to oneself, "Do we want this lung ever to expand after pneumothorax?" Would it not be much safer, instead of carrying on pneumothorax for 5 years or even a longer period of time and then letting that lung come up—would it not be much wiser rather than subjecting that patient to several years' duration of pneumothorax, to

do a thoracoplasty at once instead of playing about with pneumothorax and cutting adhesions, with the dangers of both? I believe that it is not a simple operation.

One will get 100 per cent perfect results if one selects only certain cases, but if one takes the run-of-mine, one must expect trouble. At first one should be timid and cut only strings and membranes that can be transilluminated. I believe the short adhesions practically all contain lung tissue.

Now, because one cuts across tissue, containing lung, it is by no means certain that one is going to get into trouble. But adhesions are formed by infection. Adhesions are all potentially infective by nature. One cannot see microscopic tubercles within adhesions; one can only see those tubercles on the surface, and every time one cuts across a thick adhesion, there may be hidden tubercles.

In regard to technic, I have been playing around with many types of instruments, the Chandler, the Unverricht modification of the Jacobaeus and the Mauer, and I am using now a combination of the three. I am using the Unverricht and the angulated vision scope for general inspection; and a Chandler direct scope for forward-looking vision. It has a fine little cutting electrode in it. It can also take a needle for direct anesthetization of adhesions. I feel, after using both the unimanual and the bimanual instruments that the bimanual is best. While at first it is clumsy, one gets, after a little experience, quite dextrous, especially with the curved cautery. One can reach any part of the hemithorax with it.

I do not think the perfect thoracoscope has been invented yet. It will come in time.

In regard to my silver clips, I have used them on only two patients. I am not advocating them for general use, as I said in my paper. I do not think they are universally applicable. They have not done any harm. Those patients are both alive now, and they are quite well.

I do not like to say a great deal about the open method. I am familiar with Dr. O'Brien's work, and he, with that operation has probably got better results than anyone else. But I feel, without making a general statement, that if an adhesion cannot be cut safely with a so-called closed method, it should not be cut at all. That is a general statement. If I were shown a particular case, with bilateral cavities necessitating bilateral compression, I might not be so positive about it.

DR. E. N. PACKARD (SARANAC LAKE, N. Y.).—I am quite out of my depth in this discussion. I am only a humble clinician who has nothing to do with the technical end of the procedure.

There are two points, however, that are interesting from the clinician's standpoint. One is that it is quite impossible to tell from the x-ray alone whether adhesions can be cut or not. In some x-rays one sees what appears to be extensive adhesions and yet the surgeon finds they can be cut quite readily. In other cases where adhesions seem from the x-ray standpoint to be string-like affairs, the operator may find extensive involvement, not suitable for cutting. The x-ray, then, is not a wholly reliable means of judging the number, or type, of adhesions present.

The other point is that in some cases in which, from the surgeon's standpoint, the pneumolysis was technically unsuccessful, clinically we have obtained excellent results.

DR. JOHN ALEXANDER (ANN ARBOR).—You will be interested to know that the cannulas of the present model of the Jacobaeus-Unverricht instrument set are of such size that the thoracoscope or the straight cautery rod may be introduced in either cannula.

May I take this occasion to correct an unintentional error that Dr. Bethune made in an intrapleural pneumolysis article in the February, 1933, issue of the JOURNAL OF THORACIC SURGERY? He stated that "Matson, Alexander, and O'Brien

have had nonfatal hemorrhages occur with loss of from one thousand to two thousand cubic centimeters of blood.'' I have had no hemorrhage whatever in my total of approximately 60 cases. Dr. Bethune would, of course, have gladly made this correction if I had had an opportunity to invite his attention to the error.

DR. JULIAN A. MOORE (closing).—I was hoping that somebody would show how to make this operation an easy one. Apparently all of us are having the same difficulty. It is not an easy operation. In regard to the indications of the operation, I do not believe pneumolysis should be attempted until pneumothorax has been continued from three to six months. Certainly there is a large percentage of adhesions that will stretch sufficiently with negative pressure, if given time. If, after a reasonable trial, cavities are still open or the sputum contains tubercle bacilli, the operation of pneumolysis should be considered, provided the adhesions are suitable. I see no reason for continuing an unsatisfactory pneumothorax over a long period of time, and I do not believe that it is safe to use positive pressures in order to stretch adhesions. I am sure that the vast majority of men who use pneumothorax therapy would condemn the use of positive pressures to stretch adhesions.

Dr. O'Brien has reported the largest series of open pneumolysis. It appears to me that his technic may prove to be the ideal technic for the open method, if he will permit me to still call it an open method. However, Carter's technic of stripping the adhesion from the parietal pleura seems to insure against any danger of injury to the lung, which, to my mind, is the critical point of either operation. I do not see how, through such a small incision as Dr. O'Brien makes, he can tell whether the adhesion contains lung tissue more readily than one can by using the thoracoscope. However, I am speaking without personal experience with this operation, as he has been speaking without experience with the thoracoscope.

I agree with Dr. Bethune that it is often hard to decide whether thoracoplasty or pneumolysis should be done on a given patient. Many factors would influence one in making his decision. With my limited experience I would hesitate to attempt the division of doubtful adhesions, and yet I have been so impressed with the work of Maurer in this operation that I believe a surgeon with a larger experience and with the adoption of his technic could greatly extend the indications for pneumolysis. His articles are very instructive to any one who is interested in this work, and it seems to me that he has struck the keynote of the technic in being able to avoid injury to the lung. However, his technic requires a great deal more skill than dividing the adhesion in its center. I find the execution of his technic extremely difficult.

It is my personal opinion that the Unverricht thoracoscope is the best that is available on the market. I am satisfied that certain of the accessory instruments would be quite useful if one can afford them, particularly Kremer's direct vision telescope. Unfortunately, most of us have to limit ourselves to one instrument, and I would advise any one who buys a thoracoscope to buy the Unverricht scope, which the majority of operators use.

I wish to congratulate Dr. Drash on the excellent results that he has reported at this meeting. His results are extremely good and certainly better than most operators have obtained.

I agree with Dr. Fisher that one can avoid hemorrhage with the galvanocautery, but I am satisfied that the electrosurgical method eliminates most of the disadvantages of the galvanocautery.

A NEW PRINCIPLE OF PULMONARY COLLAPSE WITH PRODUCTION OF EXTREME ATROPHY AND CIRRHOSIS OF THE LUNG—AN EXPERIMENTAL STUDY*†

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THE methods in common use for producing pulmonary collapse in the treatment of diseases of the lungs with cavitation are not entirely satisfactory, largely because the degree of compression obtained is sometimes insufficient to obliterate the cavities. Excavations with soft walls become obliterated with relaxation of the involved lung, but those with stiffer walls require not only complete relaxation but also compression of the lung—often much more forceful compression than can be obtained therapeutically.‡

In all compressive measures in clinical use, the force of compression is ordinarily transmitted in like measure against the corresponding side of the mediastinum, so that the compression is necessarily limited in degree to the small amount of positive pressure which the mediastinum is able to bear unilaterally without cardiorespiratory embarrassment.

Pneumothorax is the only procedure which (when no pleural adhesions exist) can regularly be used to compress the lung, even to this limited degree. Paravertebral extrapleural thoracoplasty usually does not even completely relax the lung, as is evidenced by the fact that the intrapleural pressure on the treated side usually remains somewhat negative[2]; but compression is obtained regularly if the collapsed lung is markedly swollen.§ When anterolateral rib resection is combined with paravertebral thoracoplasty, compression is more frequently but not always obtained. Large plombe, especially those given in combination with

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‡The present use of the terms relaxation, compression, and collapse in relation to the lung needs to be explained. Pulmonary relaxation is that state of the lung wherein the organ is reduced in size from its own elastic recoil following release from the expanding influence of the chest wall. The degree of the relaxation is proportional to the degree of the release. The discovery of any air in the alveoli of the affected lung rules out complete relaxation, since the unhindered elastic recoil is capable of quickly eliminating all the air. Pulmonary compression is the state of the lung residing in an environment of positive (supra-atmospheric) pressure, and can exist only in the presence of complete pulmonary relaxation. (While air is in the lung, that organ pulls away from the chest wall and produces an environment of negative pressure.) Thus, the discovery of any air in the affected lung also rules out compression. Pulmonary collapse is used to mean any degree of pulmonary relaxation alone or pulmonary relaxation and compression combined.

§The volumetric relationships between the inflamed airless lung and the uninflamed airless lung have been studied and set forth in another article.[3]

thoracoplasty, commonly cause some compression. The compression from either thoracoplasty or plombe is exerted entirely or mainly in only one direction, i.e., from the side, rather than in all directions, as in pneumothorax.

Several experimenters[4, 5, 6] succeeded in increasing appreciably the stability of the mediastinum by injecting various foreign substances into the connective tissue of that septum. The effect was obtained either immediately from the bulk of the substances themselves, or subsequently from the fibrosis engendered. The method may quite possibly be used beneficially in patients to heighten the amount of pulmonary compression obtainable with the standard collapse measures (which was the purpose entertained); but the theoretical objection that the pressure and irritation of the injected substances may result eventually in damage to the pliable cardiac auricles and large veins and the sensitive nerve plexuses of the mediastinum, presents itself.

The experiments to be described were made in dogs to test a new principle of pulmonary collapse, from which might be developed an improved therapeutic method. The chief advantages would be forceful compression of the lung independent of mediastinal stability, and greater selectivity and uniformity of compression. A thin soft envelope of foreign material was wrapped loosely about one part of the lung, in order to bring about the development of a fibrotic capsule in the visceral pleura of the enwrapped part which would contract circumferentially and compress the part.

EXPERIMENTAL METHOD

Twenty dogs of nearly uniform weight were used. Four of these were expended primarily in the elaboration of technic, and the remainder formed the basis of this report. In these, the right side of the chest was opened, the lower lobe was inclosed in a rubber envelope, and the chest was closed. The dogs were killed after periods varying from four days to seven months, and autopsies were performed.

Technical Details.—After a preliminary injection of morphine, each dog was anesthetized with ether and prepared for a sterile operation. The right pleural cavity was opened by a 10 cm. incision in the sixth intercostal space, and positive pressure breathing was started. The mediastinal ligament of the lower lobe was divided so as to deliver the lobe. A sterilized envelope, which had been made in the shape and size of the lobe by sewing together pieces of well-washed surgeons' gloves, was drawn over the lobe, adjusted, and sewed so as to cover all its surface, up to the point of entry of the bronchus, artery and vein to the lobe, with special care to avoid compression of the lobe or its hilic vessels (Figs. 1 and 2*A*). Pneumothorax was then excluded by inflating all lobes, and the wound in the chest wall was sutured. The anesthesia was stopped.

The postoperative symptoms were carefully watched, and frequent estimations of the red and white cells were made. Roentgenograms of the chest were taken every few days for the first month and less often thereafter. Whenever dyspnea developed, a needle was inserted into the pleural space and fluid was searched for and, if found, was removed. The fluid so obtained was measured and examined histologically and

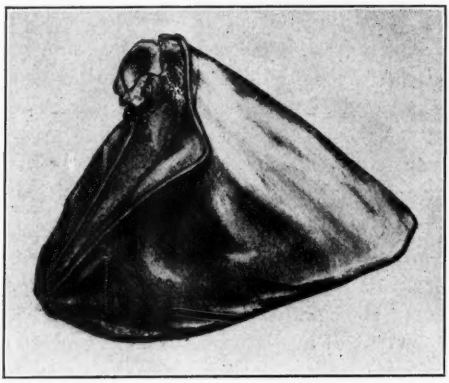
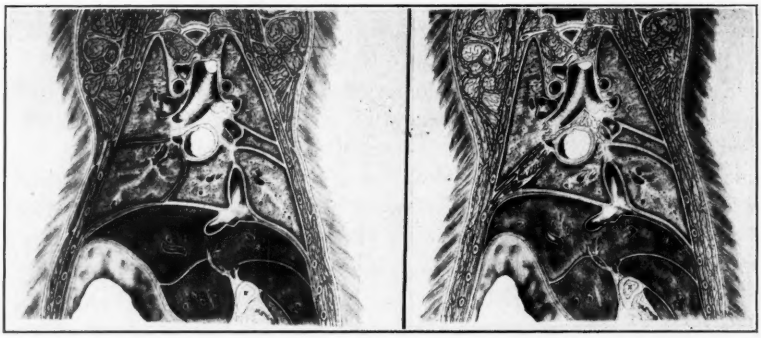


Fig. 1.—Plaster model of the right lower pulmonary lobe with the rubber envelope covering it, showing the completeness and looseness of the covering. In practice, the slit is sewn shut after the application.



A. *B.*

Fig. 2.—Frontal sections of two dogs' chests, in the same plane, showing the visceral relationships. *A*, Just after the envelope (heavy black line) was applied, when all organs were in normal position. *B*, At five months, when the treated lobe was atrophied and retracted, the envelope was crumpled about the lobe, the adjacent lobes were hypertrophied, and the heart, diaphragm, and chest wall were displaced somewhat toward the affected lobe. (Compare *B* with Fig. 3.)

bacteriologically. One or two dogs were killed and examined by autopsy at the end of each of the following periods after operation: four, seven and twelve days; two, three and five weeks; and five and seven months. Gross and microscopic examinations were then made of the lungs, particularly of the right lower pulmonary lobe.

RESULTS

Clinical Behavior.—The immediate effects of the operation were severe. The dogs were inactive, ate little or none at all, lost considerable weight, and developed moderate anemia and leucocytosis. In the majority of cases, large quantities of exudate accumulated in the right pleural cavity, the mediastinum was displaced extensively to the left, and moderate to marked dyspnea developed, reaching a maximum between the fourth and the tenth days. In the others, exudate accumulated in smaller quantities, the mediastinum was only slightly displaced, and dyspnea was

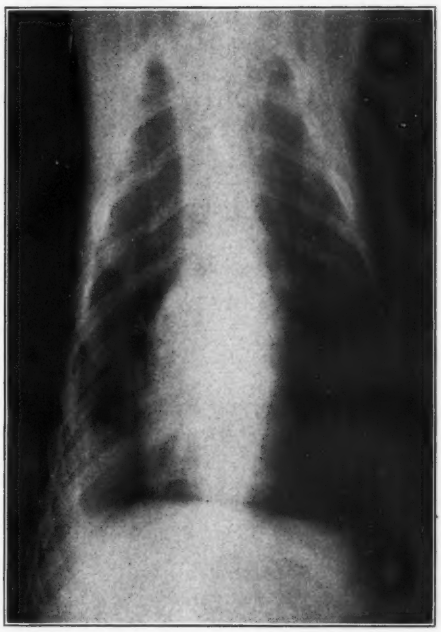


Fig. 3.—Roentgenogram at five months, showing the consolidation formed in the lower right lung field by the treated lobe and its envelope, the slight shift of the heart and right hemidiaphragm, and the flattening of the right thoracic wall.

slight or entirely absent. Aspiration was performed in the first group only, one to four times between the fourth and twelfth days, each time obtaining 300 to 400 c.c. of deeply sanguineous fluid. The fluid was sterile in eight dogs and contaminated in four. The dyspnea was considerably relieved by aspiration—permanently in those with sterile fluid and temporarily in those with contamination. The latter dogs died six to twelve days after operation. Two other dogs developed distemper shortly after the twelfth day and were killed immediately (in order to prevent this disease from changing the pathologic processes related to the surgical operation). No other respiratory symptoms appeared. About twelve

days after the operation the surviving dogs became more active, and soon thereafter they began to behave normally and recovered their previous weight and blood condition. The pleural fluid gradually diminished and, in those which were allowed to live long enough, it seemed (by x-ray examination) to disappear completely. At the fifth month, roentgenographic examination showed the entire right hemithorax to be

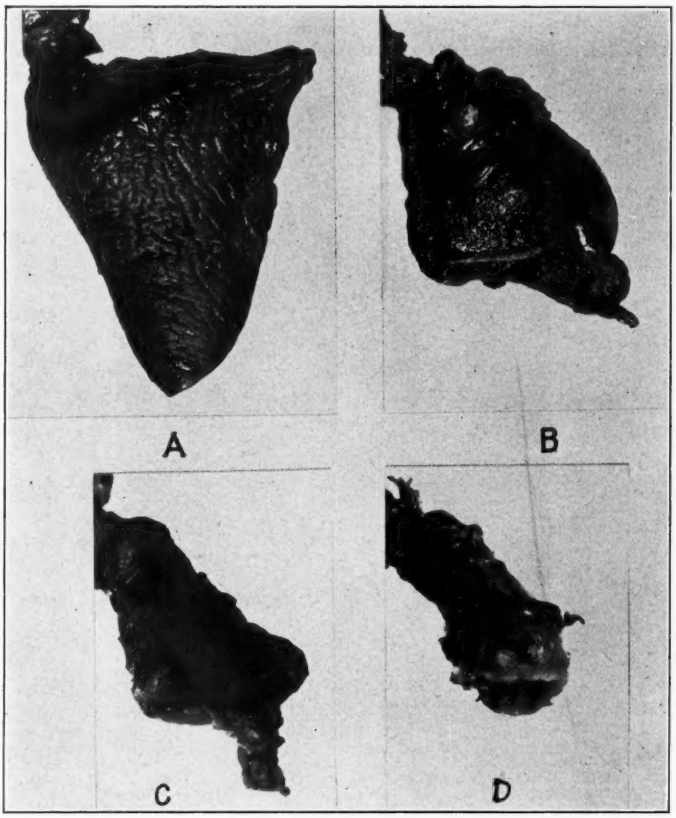


Fig. 4.—The treated lobes of four dogs, showing different stages of collapse (envelope removed). *A*, Four days after operation when relaxation of the lobe was complete but compression was not begun. The fibrin was peeled off to disclose the wrinkled surface and normal angular shape of the lobe. *B*, At two weeks, when the fibrous capsule was well formed (see also Fig. 6) and exerting marked compression, and the lobe was rounded and reduced in size. *C*, At five weeks, when the capsule was exerting further compression, and the lobe was more rounded and reduced. *D*, At five months, when the compression was complete and the lobe was a mere nubbin of fibrohyaline substance.

somewhat flattened, the ribs and hemidiaphragm on the same side limited in motion, the right hemidiaphragm slightly elevated, the heart drawn a little to the right, and the lowest one-fourth of the right lung field consolidated (Fig. 3).

Gross Postmortem Findings.—On the fourth day, the right pleural cavity was occupied by considerable quantities of serosanguineous fluid, and masses of shaggy fibrin clung to both the visceral and the parietal pleura throughout that side of the chest. The exudate was maximal in amount at the twelfth day. In some cases it filled the hemithorax completely and displaced the mediastinum to the opposite side, while in others it totally filled only the lower part of the pleural space and semi-filled the upper part. After that, the exudate decreased steadily in amount, since at five weeks there remained very little and at five months there was practically none. The last vestiges resided wholly at the base of the chest.

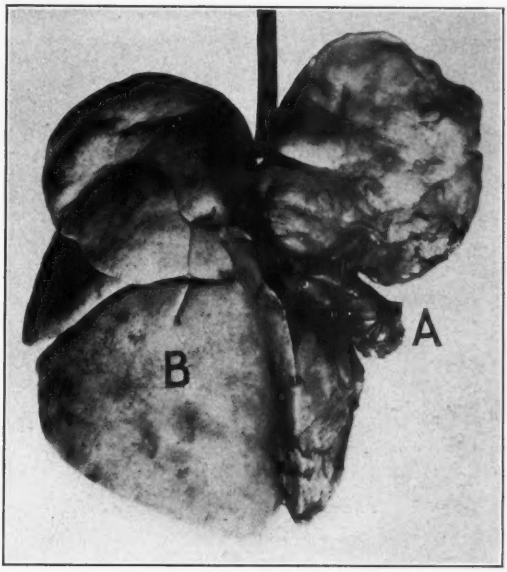


Fig. 5.—The lungs at five months, inflated as much as in life; demonstrating the extremely small size of the treated lobe, *A*, compared with the opposite lower lobe, *B* (normally of equal size). Also, the fully air-containing condition of the other lobes on the side of the treated lobe is indicated.

On the fourth, seventh, and twelfth days, the right lower lobe was always atelectatic, as from simple relaxation. Thus, it was small, airless, flabby, wrinkled and purple brown (Fig. 4*A*). The other lobes on this side were in the same condition in the animals with the more extensive exudation, but they were only partially collapsed in the cases with the smaller amounts of exudate. Beginning with the fourteenth day, when the fluid started to disappear, the right lower lobe always differed from the other lobes on the same side in that the former became steadily smaller and changed its normal angular shape for a rounded one, and the latter became gradually larger from inflation and maintained the angular

shape. Indeed, the contour of the lower lobe was distinctly rounded as early as the twelfth day (Fig. 4*B*). By then, also, the exudate on that lobe could not be peeled off to expose the pleura, as before, and it showed a distinct fibrous quality next to the pleura. The cut surface of the lobe had the dark purple brown color of atelectasis, but it was moist and greatly bulging, unlike atelectasis due to simple relaxation. By the fifth week, the lobe was still smaller and more rounded—much less than one-half the size the lobe would be if atelectatic from simple relaxation, and roughly cylindrical (Fig. 4*C*). A heavy fibrous capsule surmounted by shaggy fibrin covered all parts of the organ, and the consistency of the parenchyma was much firmer than before. The cut surface of the lobe was flat and red gray. All other surfaces in contact with the rubber

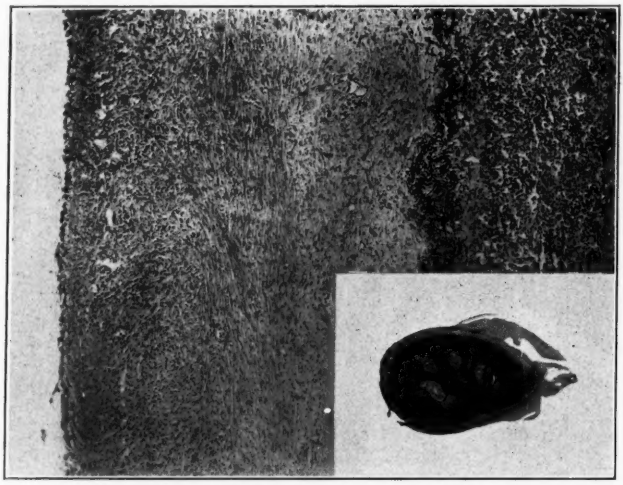


Fig. 6.—Section (×55) of the cortex of a treated lobe at two weeks, showing the fibrous pleural capsule and the adjacent compressed parenchyma. Insert: Entire cross-section (natural size) of the same lobe, showing the proportions of the capsule and the density of the parenchyma.

looked just like the one of the lower lobe, but the other lobes were nearly or wholly expanded, intimately adherent to the chest wall, air containing, and without gross parenchymatous changes. At five to seven months, the lower lobe was considerably firmer and smaller than before—about the size and shape of the two terminal segments of a man's middle finger (Fig. 4*D* and Fig. 5). It was covered with a gray fibrous coat, 1 mm. to 2 mm. thick; and the cut surface of the parenchyma was uniformly gray and structureless. A few large, completely obliterated bronchi could be seen in the central and hilic regions.

The envelope was always found to be in position surrounding the right lower lobe and was bathed inside and out with pleural fluid. At the fourth day, the envelope was already greatly collapsed; and at sub-

sequent stages of examination it was collapsed more and more, keeping pace with the reduction in size of the lower lobe. Also, at each stage, the pleural compartment occupied by the envelope was smaller, in pace with the expansion of the other lobes on this side. Finally, the much crumpled envelope containing the small fibrous nubbin which represented the lobe lay in a narrow space situated on the diaphragm and extending from the costodiaphragmatic sulcus to the pulmonary hilus.

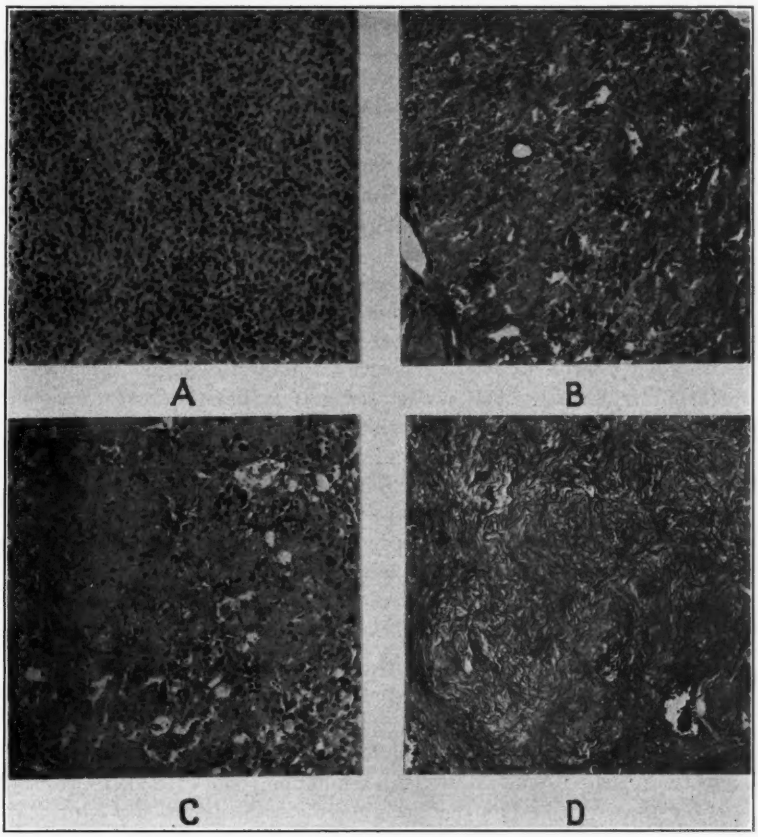


Fig. 7.—Sections (×155) of the parenchyma of the treated lobes of four dogs, at different stages of collapse. *A*, One week after operation when relaxation was complete, compression was beginning, the alveoli were totally collapsed, the tissue cells were closely crowded, and the capillaries were dilated. *B*, At three weeks, when cirrhosis (interstitial fibrosis) was advanced and general vascular dilatation was present. *C*, At five weeks, when cirrhosis was further progressed and hyalinization was beginning. *D*, At five months, when compression was complete and the parenchyma was totally transformed into collagenous and hyaline tissue.

Microscopic Findings.—On the fourth day, the pleura of the right lower lobe showed marked vascular dilatation and a thick covering of fibrinosanguineous exudate. By the seventh day, fibroblasts and capil-

laries from the pleura were invading the exudate to some extent; and this process of organization was far advanced by the fourteenth day. At five weeks, there was a thick layer of mature, densely packed, fibrous tissue on the lobe (Fig. 6); and at five to seven months, this capsule was almost wholly hyalinized. The pleural surfaces of the other lobe and of the chest wall, where they touched the envelope, showed the same capsule formation.

The parenchyma of the right lower lobe had, on the fourth day, the appearances of acute atelectasis, except that some of the alveoli, instead of being fully collapsed like the rest, were partially collapsed and contained large phagocytic cells. On the seventh day, the tissues gave the appearance of still further condensation, for the nuclei of the structural cells were more closely packed than before (Fig. 7*A*). The capillaries were everywhere dilated, and there were a few small foci of erythrocytic extravasation beneath the pleura. Two weeks after the operation, many young fibroblasts were found in all regions, apparently springing from the pulmonary connective tissues, and the structural cells were much thinned out (Fig. 7*B*). At three weeks, fibroblasts were the dominating cells, and they were beginning to hyalinize. Indeed, these cells had entirely replaced the epithelial and muscular elements of the alveoli and smaller bronchioles (Fig. 7*C*). At five to seven months, the process of fibrous replacement had progressed so far that the lung consisted merely of interlacing collagenous and hyaline fibers (Fig. 7*D*), with a few large bronchi which were intact but held collapsed in the scar tissue. The large vein at the hilus was partially collapsed, but the accompanying artery was fully patent, and the large veins and all small vessels throughout the lobe were somewhat dilated. At no time in the course of the process was there evidence of either inflammation or necrosis.

DISCUSSION AND CONCLUSIONS

The initial relaxation of the lobes on the right side was obviously due simply to the massive pleural exudate which developed from the beginning in reaction to the presence of the foreign body. And the reinflation of all these lobes except the lower one resulted merely from the resorption of the exudate. In their reinflation, they extended not only into the space which they had originally occupied but also into that which was no longer occupied by the lower lobe; and it was probably on this account that the heart and diaphragm were saved from becoming extensively displaced to the right and upward respectively after the resorption of the pleural exudate, in compensation for the reduction of size of the treated lobe. This lobe failed to re-expand with the others, quite obviously because of the fibrous capsule which had begun to develop in its pleura. The capsule doubtless represented the reaction on the part of the pleura to the foreign body which surrounded it. Vigorous contraction on the part of the capsule was distinctly in evidence as early

as the twelfth day, both from the marked rounding of the lobar shape and from the great bulging which the parenchyma exhibited when the lobe was transected for examination. There was, of course, some hydrostatic pressure upon the lobe in vivo while the fluid lasted, but that pressure could not have been responsible for these two phenomena. The atrophy of the lobe came on so rapidly that disuse could scarcely have been the cause; the capsular pressure was more likely the cause. Although the pressure was evidently very great, it was never great enough to produce necrosis. Compared with either the fully inflated lobe or the fully relaxed one, the mass of tissue which remained at five to seven months was exceedingly small.

That a thick and powerfully contracting fibrous capsule would develop in the visceral pleura from the presence of the foreign body and that the lobe would become atrophic from the pressure were expected beforehand; but, that cirrhosis would develop was not anticipated, nor is the cause for this alteration yet known for certain. Two explanations for the cirrhosis have been considered. One was suggested by the fibrosis of the muscles of an extremity which takes place under a too tightly applied plaster cast (Volkmann's ischemic contracture). This explanation seems the less likely, because the fibrosis in the muscles is thought to be caused essentially by the ischemia, whereas in the case of the lung lobe no ischemia was found but rather a distinct hyperemia at all stages. The other factor entertained was the hyperemia itself, since chronic passive congestion of the lung in heart failure is ordinarily followed by parenchymatous fibrosis. The large phagocytic cells in the alveoli recalled to mind "heart-failure cells." Of course, the hyperemia was chronic. It was evidently passive in type and obstructive in origin; for that part of the fibrous capsule which reached to the hilus was found to press upon and partially to collapse the hilic vein but not the hilic artery. (It is reasonable to expect that the vein would have been compressed first on account of its lower internal tension). Furthermore, experiments of Smirnoff,[7] in dogs, showed that chronic obstructive hyperemia from ligation of the vein of a lung lobe leads to parenchymatous fibrosis. However, the degree of the fibrosis in both heart failure and ligation is very much less than that which occurred in these experiments, so there is probably some other, unknown, associated cause.

The method has so far met the expectations, but before it can be applied to man with a reasonable degree of safety, it requires further study of the significance of the parenchymatous changes produced and further technical elaboration. Doubtless the compression will be helpful in obliterating pulmonary cavities, but the question is whether or not the cirrhosis will be beneficial. If the cirrhosis represents an exuberance of vitality on the part of the connective tissues resulting from some stimulation, as it seems to, it should be a great aid in walling off and destroying lesions; but, if it is merely a reparative response to injury

inflicted upon the parenchyma directly or indirectly by the operation, the treatment as a whole should be detrimental to the pulmonary healing processes. Moreover, the postoperative reaction seemed too severe for safety in patients already toxic from pulmonary disease. Search for a less irritating material than rubber is in progress. Also, a two-stage operation is being tried, wherein the first stage serves to bring about obliteration of the upper half of the pleural cavity, to reduce the amount of exudation and dyspnea following the application of the envelope at the second stage. Furthermore, a smaller envelope is being tried and the lobe is being left relaxed, rather than expanded. And it is planned to determine the earliest time after application of the envelope at which this foreign body can be removed without jeopardizing the fulfillment of the compressive effect.

The experiments are described at this time less on account of the possible clinical application of the method, than because of the theoretical interest of the process itself which they portray. It is surely a strange circumstance that a part of the lungs may be crushed practically out of existence by the contraction of its fibrotic pleura. The completeness with which the contracting capsule covered the lobe was undoubtedly essential, since, of course, the contraction had to be from all sides in order that the lobe might be compressed. Thus, such atrophy and cirrhosis as this usually fail to appear in total unilateral empyema in man, probably because the collapsed lung in this case falls immediately against the mediastinum and becomes adherent thereto, so that only the lateral aspect of the lung is exposed to the action of the bacterial irritant and the formation of a fibrous capsule, and circumferential contraction and compression are avoided. Moreover, in these experiments, the contraction would undoubtedly have been greatly interfered with if provision had not been made for permanent isolation of the lobe from the ribs and other relatively immobile structures in the environment, by covering the lobe with a material impervious to fibroblastic invasion. Thus, compression and its effects fail to occur in the tuberculous lung which develops spontaneously a fibrotic pleural thickening, without separation from the chest wall. This is obviously because the lung is adherent to ribs which resist the contraction, so much that the resulting reduction of pulmonary size is usually insufficient even to cause complete airlessness.

SUMMARY

Experiments involving 20 dogs were performed to test on normal lungs a new method whereby one portion of the organ was afforded very vigorous compression with such reduction in size and such cirrhotic change as to render it virtually extinct by causing its pleura to thicken and shrink.

The lobe was inclosed as far as the hilus in a loosely fitting envelope of thin rubber and then was left fully inflated. The foreign body produced

an intense pleural reaction, so that free fluid accumulated and the pleura in contact with the rubber became thickened and fibrous. The fluid collapsed the lobe and soon afterward underwent absorption, while the fibrous capsule of the lobe maintained and furthered the collapse by shrinking progressively. The envelope relaxed in folds about the organ. Hyperemia and fibroblastic proliferation developed in all parts of the parenchyma; and the cirrhosis progressed until all respiratory structures except the largest bronchi were replaced with scar tissue. These tubes remained intact, although collapsed. The shrinkage was so extensive as to reduce the lobe in a few months to a very small firm mass. The space so left was filled mainly by the neighboring lobes, which were hypertrophied, and otherwise by the heart, hemidiaphragm, and chest wall, which were slightly displaced.

The pulmonary compression obtained by this method is unique because of its independence of mediastinal stability, as well as because of its superior forcefulness. It should have special advantage when used clinically for collapsing stiff-walled cavities of the lung. Also, the cirrhosis may possibly prove advantageous for limiting and healing noncavernous lesions. However, further study of the meaning of the parenchymatous changes and much technical refinement are needed before application is made to man.

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DISCUSSION

DR. NORMAN BETHUNE (MONTREAL).—I understood from Dr. Van Allen that he did not constrict the root of this lobe.

I should like to report, apropos of the use of these rubber bags to enclose a lobe, on the use of a rubber bag in a lobectomy that I did last week which might be of some significance at this time. A typical left lower lobe lobectomy was to be done, and we had the idea to enclose that lobe in a bag which was made especially for us by the Sterling Rubber Company, makers of surgical rubber gloves, to prevent infection of the pleural cavity.

(Illustrating.) We planned to put the lobe in the bag in this fashion, and then to constrict it at the hilus, let our lobe rot off inside the bag, and having brought

the end of the bag out through the skin, we would prevent or limit infection inside this pleural space.

This was what happened. The lung was adherent firmly to the diaphragm. The electrocautery was used to cut across the adhesions, with the Cameron light in the usual fashion behind them to transilluminate. One or two of the adhesions were pretty tough and needed clamps and ties.

When the lobe was entirely free, about the size of my fist, I pulled the rubber bag over, like that, and ran it up to the hilus. This was very high. I should think that probably at least 95 per cent of the lower lobe was in the bag. I then put on a tourniquet, around here, and tightened it and brought it out through the wound. We had the idea that day by day we would tighten the tourniquet and so get a dry gangrene in the lobe, and finally we would have it entirely choked off, and then would be able to pull the whole thing out.

This is what actually happened: The man died in thirty-six hours, and he died from a combination of three causes. He died in a state of shock. First of all, he had anuria. He passed only four ounces of urine in thirty-six hours, complaining of great pain in the kidneys. There had been pus in his urine for several months before. It seemed to be a kidney "strike." There was also a small tear in the bag, and we found blood to be oozing out of the denuded surface and leaking out through the tear. The amount of blood lost was not sufficient to cause his death; I am convinced of that. He had been given 3000 c.c. of intravenous saline in the thirty-six hours and an additional 450 c.c. of whole blood. The other cause was possibly histamine absorption.

At postmortem the lobe was very voluminous, hard and congested. As soon as I saw it I suspected at least one of the contributing factors to his death.

I injected with lipiodol the pulmonary artery on the left side and then injected the bronchi. The upper lobe was very nicely injected with lipiodol, and by x-ray examination I found that a branch of the pulmonary artery running down to the lower lobe in the middle of the root was uncompressed. The bronchus to the lower lobe was entirely constricted. The man had been raising twenty ounces of sputum daily for the last six years. He raised no sputum or had no cough for the thirty-six hours following the operation.

We had the bronchus constricted but protected by the cartilaginous plaques; in the center of the constricted root was a small uncompressed branch of the pulmonary artery. I blame the voluminous bag plus the cartilaginous plaques in giving it too much bulk to constrict with the tourniquet.

DR. POL. N. CORYLLOS (NEW YORK).—It is always a great pleasure for me to discuss a paper of Dr. Van Allen's. We work along similar lines, and if we do not always agree, we derive a mutual benefit from our discussions.

His presentation is a valuable piece of experimental work on extreme compression and cirrhosis of the lung. He believes that the shrinkage and atelectasis which followed were due to compression exerted by the fibrosis which developed under these conditions.

I am not ready to agree with this interpretation. In fact the lobe enclosed in the rubber bag was already atelectatic in the animal sacrificed only five days after the experiment, which was certainly too early for the development of fibrosis. Second, in the illustrations projected we do not see very marked fibrosis from the periphery to the center of the affected lobe but a diffuse process of fibrosis. Third, the lumina of even larger bronchi are compressed, which is not found in fibrotic interstitial pneumonitis, to which Corrigan gave the name of cirrhosis and which so often is followed by bronchiectasis. In cirrhosis of the lung dilation of the bronchi and not compression is what generally occurs.

For these reasons I think that the succession of phenomena is somewhat different. Following thoracotomy and manipulation of the lung serous exudate developed as it is the rule. This exudate was gradually absorbed and the lung reexpanded. This could not take place, however, around the lower lobe which was enclosed in the rubber bag; this lung remained compressed and became atelectatic very rapidly, as it is shown in the five days' specimen. Persistent atelectasis on one hand and thickening of the visceral pleura on the other hand lead to fibrosis because of persistent anoxemia; this is true for all tissues as shown by Leriche and Policard and for the lung more especially, as I have tried to show recently.

It is by the same mechanism, I believe, that cirrhosis of the lung follows chronic empyema, pneumothorax prolonged for several years, or thoracoplasties.

It should be made clear, however, that definite fibrosis of the lung parenchyma by prolonged compression is difficult when the lung is healthy. In chronic empyema or prolonged pneumothorax reexpansion is possible following decortication of the lung. This seems to be due to the dual circulation of the lung, functional (pulmonary artery and veins) and nutrient (bronchial arteries and veins), which overlap functionally in a small part. Atelectasis disrupts the functional circulation but affects very little the bronchial artery-vein system.

It is not the same when the lung parenchyma was diseased before its collapse and lesions of the small bronchial vessels were present. In these cases following compressive atelectasis, which obliterates the functional circulation, necrosis or fibrosis of the affected areas will follow, because of excessive ischemia, regulated by the degree and the extent of these vascular lesions. Therefore in tuberculosis, for instance, following pneumothorax, fibrosis is limited to the diseased areas whereas the healthy portions of the lung do reexpand.

DR. HOWARD LILIENTHAL (NEW YORK CITY).—Many years ago in a patient in whom I intended to perform a right lower lobectomy, I loosened the lung from its adhesions to the chest wall and lightly wound a piece of rubber dam—not rubber tissue—around it, in preparation for the next stage of the operation, but the man died of an infection produced, I fancy, by the loosening up of the adhesions, which were probably not perfectly clean.

Since then I have not employed this method at all. Of course, it is obvious that Dr. Bethune's idea is a totally different thing from Dr. Van Allen's.

In chronic empyema in which the lung has been pressed into the spinal gutter until there is hardly any air left, I have been able to mobilize and reinflate by cutting through the tough although thin fibrous tissue which has surrounded the pleura, and I have been able to expand to the chest wall. In other cases there is complete fibrosis and it is like that condition described by Dr. Van Allen.

I have had specimens of these lungs examined, They were fibrotic throughout. Why fibrosis occurs in some cases and not in others we do not know at present. It is a subject for investigation.

DR. LEO ELOESSER (SAN FRANCISCO).—It seems to me that Dr. Coryllos and Dr. Van Allen have spoken of two different things. In one the compression is extrinsic. The pathologic condition that produces the fibrosis and compression is extrinsic, and in the other one, intrinsic.

I should merely like to mention that Nissen enveloped the lobes in silk preparatory to lobectomy, and has published a number of cases. He used silk—not a rubber envelope—in a similar way to that of Dr. Van Allen's. Whether he is still using that method preparatory to lobectomy as a first stage, I do not know.

DR. C. M. VAN ALLEN (PEIPING, CHINA).—The principle involved in the use of the rubber envelope in the case just described by Dr. Bethune is quite different from that in our experiments; for he employed the envelope to limit the field of

contamination about a ligated and lifeless pulmonary lobe. Nissen, Harvey and others have applied bags to the lungs for purposes similar to Dr. Bethune's.

Dr. Coryllos' suggestion, that the cirrhosis may have been due to anoxemia, is apparently based on the circumstances that pulmonary fibrosis often develops with chronic atelectasis in man and that the blood is not aerated in atelectatic lobes. This explanation has certain defects. First, the atelectatic lobe has a supply of aerated blood from the bronchial artery, any initial deficiency in which is probably very soon compensated for by dilatation of this artery. Dr. Holman showed very well that the bronchial artery is capable of rapid hypertrophy when an increased blood supply was required. Second, pulmonary fibrosis often fails to develop with chronic atelectasis, in both dog and man. Dr. Adams found that dogs' lobes, which had been rendered atelectatic for long periods by having had their stem bronchi obliterated from within, failed to show fibrosis. And when artificial pneumothorax is used in man producing atelectasis of one whole lung in which only a portion is tuberculous, the nondiseased portion usually retains its ability to reinflate totally whenever the treatment is abandoned, which it would not do if it were extensively cirrhotic. Therefore, there must be some other cause for the fibrosis which sometimes accompanies chronic atelectasis in man. It is probably the infection associated with the atelectasis.

I did not make it clear that there was free communication for the passage of fluids between the interior and the exterior of the envelope, by way of the loosely fitting neck and the interstices of the stitched seams. Because of the communication, the fluid pressure within the envelope could not have been greater than that outside of it. This would seem to answer Dr. Coryllos' query as to whether the compression of the treated lobe may have been caused by a retention of fluid under pressure within the envelope, rather than by the contraction of the fibrous pleural capsule. Without doubt the body of pleural fluid as a whole did exert hydrostatic pressure upon the lobe; but if this of itself were responsible for the atrophy of that organ, then equally extreme pulmonary atrophy would occur regularly in human cases of enduring massive sterile effusion in the pleural cavity, which of course does not occur. The presence of a very forceful pressure of quite different origin was indicated by the observation made during the second week of the postoperative course that, after the lobe was removed from its fluid environment and was transected, the cut surface of the parenchyma bulged very greatly. The second source of compression could scarcely have been other than the fibrous capsule.

EXTRA-INTRAPLEURAL SUPRACLAVICULAR APICOLYSIS*†

A New Method for Collapse of Adherent Apical Pulmonary Cavities

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IN ARTIFICIAL pneumothorax therapy for pulmonary tuberculosis one very often encounters a great difficulty and at times a failure to collapse apical cavities that may be held against the chest wall by means of dense adhesions at the cupola. Apicolysis has been suggested and tried with varying degrees of success. If the extrapleural apicolysis is not done in connection with some type of plombage, the lung is likely to expand and the operation result in a failure. In many cases the cavity, thus produced by the extrapleural lysis of the apex, has been collapsed by the use of paraffin. This substance, however, is not pliable enough and often does not give a satisfactory collapse of underlying apical cavities. Frequently, it has acted as a foreign body and caused annoying complications. In order to offer a simple and easy means of collapse for such cavities, we present our procedure in the hope that it may close apical cavities without the necessity of any major procedure such as thoracoplasty. We do not propose this operation as a substitute for thoracoplasty, but as an additional conservative means of pulmonary collapse to be used especially in patients in whom there are definite contraindications for thoracoplasty. This operation may be performed independently or at one sitting when either a scaleniotomy or a phrenic neurectomy is done. It does not require a larger incision than that which is necessary for a scalenus muscle section or a phrenic nerve resection. The operation is comparatively safe when one keeps in mind the important anatomic structures in this region, namely, the carotid sheath with its contents, the transverse cervical artery, the trunk of the cervical sympathetic, the brachial plexus and the subclavian vessels.

TECHNIC

With the patient's head inclined upward at about a thirty degree angle, a transverse skin incision (see Fig. 1*A*) is made in the supraclavicular region as for phrenic neurectomy. This incision is made one inch above the clavicle along a horizontal crease line about one and three-fourths inches long. The midpoint of this incision is over the lateral border of the sternocleidomastoid muscle. The incision is deep-

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ened through the platysma which is separated by means of the blunt end of the scalpel in order to expose the subcutaneous tissues. It is deepened into the underlying pyramidal pad of fat vertically until the sheath of the scalenus anterior muscle is reached. The phrenic nerve is found lying over the scalenus muscle in or outside of the sheath. It crosses the muscle from the lateral to the medial side and extends downward, or is found on the medial aspect of the scalenus muscle. After the nerve is isolated, it is injected with procaine, clamped, cut and is avulsed in

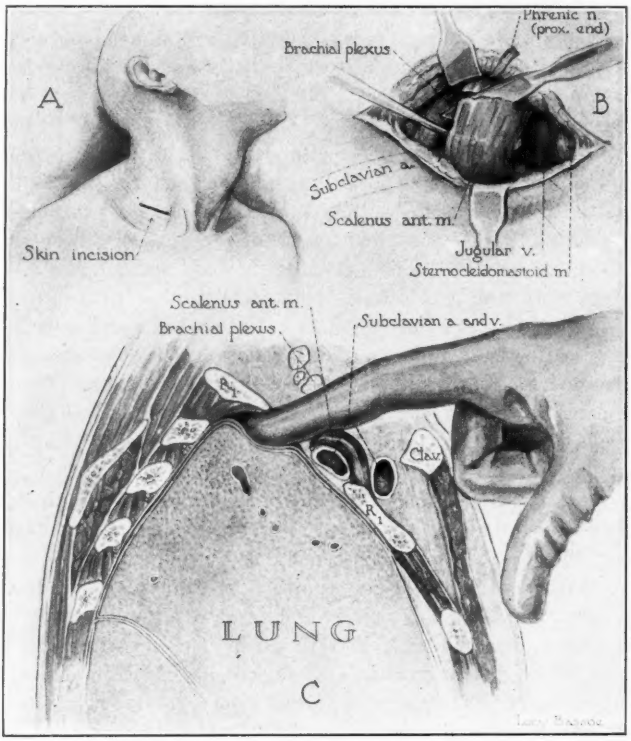

Fig. 1.—Diagrammatic sketch showing anatomic relations and steps of operation.

the same manner as in the accepted routine for phrenic neurectomy. With a Kocher dissector or with the handle of the scalpel the lateral and medial borders of the scalenus anterior are freed within the sheath. When completely freed from the neighboring structures, the muscle is cut transversely at a level as high as possible. The distal portion of the cut anterior scalenus muscle is now bent backward and downward so as to form a protective cover over the subclavian artery and thus avoid any injury to this vessel. (See Fig. 1*B*.) If it is difficult to isolate the muscle in toto without injuring the brachial plexus or the structures on

the medial border of the muscle, the sheath of the muscle may be incised and the individual fibers of the muscle may be gently torn by means of the tissue forceps or a blunt hook as high as possible, thus avoiding injury to the adjacent structures. The posterior sheath is now incised, thus exposing the underlying areolar tissue which is located over the cupola.

The index finger, bent so as to form a hook, is now inserted into this areolar tissue. The dorsum of the finger is held tightly against the transverse processes of the cervical vertebrae. The finger is pushed backward and downward, and in this way accomplishes the stripping of the parietal pleura which is found directly underneath the areolar tissue. The finger must be passed between the carotid sheath medially, the brachial plexus laterally, the vertebrae posteriorly, and the subclavicular artery anteriorly. The apex is reached and a gentle subpleural dissection is now started. It is necessary to keep the tip of the finger always close to the wall of the chest and follow the line of cleavage along the chest wall. This dissection of the parietal pleura must be extended far enough down to make certain that the area covered by the adherent underlying cavity has been completely loosened from the chest wall. The finger dissection is followed from the vertebral border medially along the mediastinal structures and then extended laterally. The dissection is then extended along the anterior wall underneath the ribs. During the dissection along the mediastinal border as it is extended anteriorly, one must keep in mind the presence of the internal mammary artery and also the thoracic duct which may be injured if one does not observe sufficient precaution. The dissection may be extended as far down as the finger can reach, always keeping in mind the necessity of following along the line of cleavage and not pushing into the loosened parietal pleura. Just below the supraclavicular fossa at the cupola one encounters irregular fibers which hold the pleura to the chest wall much like a suspensory ligament. These fibers must be stripped gently so as not to allow any tear into the pleura. The parietal pleura can be dissected as far down as the fourth rib posteriorly and the second rib anteriorly. When one has reached the pleura beyond the lower level of the apical cavity, the pleura may now be gently torn and an opening should be made large enough to convert the two cavities (intra- and extrapleural) into one cavity. If one does not desire to tear the pleura, the extrapleural space at the cupola may be filled by any one of several materials that are commonly used for a pack. The sheath of the scalenus muscle is now sutured, and the skin incision is closed with skin clips or skin sutures.

DISCUSSION

One may question the advisability and logic of a supraclavicular extra-intrapleural apicolysis on several counts. The most important of these are possible interstitial emphysema, pulmonary hernia, traumatic or in-

fectious pleurisy with effusion, tear in the lung, injury to the brachial plexus, the vagus or the sympathetic trunk, and finally, possible injury to the vessels in the neck or the subclavian vessels. The danger of interstitial emphysema is very slight unless the lung is torn and a large or persistent bronchial fistula results from the tear of the lung. With ordinary artificial pneumothorax where the intrapleural pressure is usually slightly below zero atmospheric pressure, the tendency would be for the tissues in the neck to be sucked in by the respiratory movements of the chest wall and the diaphragm. A bulge or a herniation is liable to occur only with coughing or severe straining. Even under these conditions this bulge will not be any greater than what is seen normally when the patient coughs. Within ten days there is usually sufficient amount of scar tissue at the site of the incision to prevent any bulging at the site of operation.

A pulmonary hernia is obviously out of the question for two reasons. In the first place, the artificial pneumothorax which is present will tend to keep the lung collapsed, and under such conditions the lung will show a tendency to be contracted toward the hilum and the mediastinum. In the second place, the lung may not herniate because the pivot point of the lung is at the hilum, and unless there is a marked displacement of the tissues at this point, it would be difficult for the lung to herniate.

A tear in the lung is not very likely to occur if the operator is particularly careful to follow the finger dissection along the lines of cleavage and not tear the pleura until he is certain that he is dissecting thin, virgin pleura. The feel of the lung is likely to be misleading because with the exception of definite consolidation such as in pneumonia the lung feels rather spongy and perhaps "gives" a great deal more at the site of an underlying large cavity. If, on the other hand, the lung is torn, the tendency would be for the lung to contract and a gummy fluid rich in fibrogen will produce a sufficient plug for the the vessels and bronchioles to make it air tight. In experimental work on dogs' lungs we were able invariably to close this gap by placing a slight pressure over the lung for a period of one to five minutes. The only time when such a tear would prove dangerous is when it connects directly with a larger bronchus. This prevents the collapse of the torn lung by persistently allowing air to be blown into this area. Air embolism may or may not result under these conditions. If it does, one can hope for the best and keep treating the patient for the latter condition. Details regarding the mechanics of air embolism production have been discussed in a previous paper.[1] In the absence of air embolism and in the presence of tension pneumothorax an immediate thoracotomy may be performed, and the lung sutured with the technic described in a previous paper.[2] Pleurisy with effusion may follow a tear of the lung. Such a sequela is not a serious one unless it is of the streptococcus type, which may be dangerous.

Traumatic or infectious pleurisy with effusion may follow as a result of this procedure. One certainly should not expect any more reaction than that noticed after an intrapleural pneumolysis. Moreover, an effusion in the chest cavity is nothing to be alarmed about. This occurs in from 2 to 10 per cent of all pneumothorax cases and results in no serious danger. In the presence of infectious pleurisy with effusion, repeated aspirations with or without injections of antiseptic solutions or oils will no doubt clear the pleuritic effusion. Calcium, either intramuscularly or by mouth, has been found by us to be of definite value in these cases for inducing an absorption of pleuritic effusion.[3]

The other complications are those that may usually be associated with either phrenic neurectomy or scaleniotomy. These consist of injuries to the vessels and nerves in the neck and the supraclavicular fossa and can be easily avoided if one is particularly careful about the anatomic location of these structures. It is essential to avoid as much as possible any of these structures during the dissection of the scalenus muscle and the nerve. It is for this reason that after the scalenus muscle has been reached we have made it a point to use blunt instruments all the way along except in such cases where a free and easy dissection has been made of the scalenus muscle in its sheath and we are absolutely certain that we have avoided the brachial plexus which lies laterally to the scalenus anterior muscle.

From an esthetic point of view, particularly in women, the operation will eliminate the necessity of an additional visible scar on the chest. We feel that this operation may be used in cases where the lesion is localized in the apex. In patients in whom the lower portion of the lung is comparatively healthy we feel that a conservative operation, such as this procedure, is of definite advantage over a thoracoplasty because it allows the healthy portion of the lung on the operated side to maintain the proper respiratory function. There may also be a definite indication for this operation in emergency conditions such as acute hemorrhage from the lung where it is definitely known that the hemorrhage arises from an apical cavity which is impossible to collapse by means of artificial pneumothorax.

This operation has been tried on fixed specimens in the anatomical laboratory of the University of Illinois through the courtesy of Prof. Otto F. Kampmeier. It has also been tried on fresh cadavers in the Department of Pathology of the Cook County Hospital through the courtesy of Dr. R. H. Jaffe and in dogs at the Research Laboratories of the Municipal Tuberculosis Sanitarium under the direction of Dr. H. C. Sweany. In these specimens, even though we have dealt primarily with normal thin parietal pleura, we were able to strip the pleura down to the fourth rib posteriorly and to the second rib anteriorly without producing a tear. At the cupola we encountered a strand of the suspensory fibers. These were carefully stripped along the chest wall, and thus a tear of the

redundant pleura was prevented. Because of the danger of producing a tear in the parietal pleura over the adherent cavity during the dissection, we wish to emphasize again the necessity of dissecting the pleura along the chest wall and follow the line of cleavage until the free parietal pleura is reached and dissected.

Closest to our procedure is the operation of Coffey,[4] in which the scalenus muscle, the phrenic nerve, and the long thoracic nerve are cut, thus facilitating an anterior resection of the first rib. Gale and Middleton[5] have emphasized the necessity of adding scaleniotomy to the operation of phrenic neurectomy for moderately advanced cases of pulmonary tuberculosis. They have demonstrated that after phrenic neurectomy there is an overactivity of the scaleni and intercostal muscles which may impair the desired effect of the simple phrenic nerve operation. Gregoire[6] also described a similar operation. He performed a graded thoracoplasty starting from below and resecting five ribs during the first sitting and five ribs during the second. At the third stage, in addition to removing the first and second rib, he resects all three scaleni and the phrenic nerve. He also pushes down the cupola without packing but mentions the inability of keeping the pleura down without pack. Lyonnet[7] has also reported the use of this procedure. Felix[8] states that Loeschke and Rost have performed the same operation but on too few cases to permit judgment as to the benefits of this operation. However, we have been unable to find any report which recommends a combination of phrenic neurectomy, scaleniotomy and apical pack done through a simple phrenic neurectomy incision, nor have we found such a procedure recommended as a decisive and direct attack upon apical involvement which could not be handled by any of these procedures alone but which still does not warrant a thoracoplasty.

SUMMARY

1. An operation is proposed which combines scaleniotomy, phrenic neurectomy and an extra-intrapleural supraclavicular pneumolysis so that one or more of these may be performed at one sitting.

2. The direct collapse of adherent apical cavities may be maintained either by plombage over an intact pleura, or by simply opening the parietal pleura into the extrapleural cavity to produce an extra-intrapleural pneumolysis.

3. This operation is proposed as a procedure to be used where a conservative collapse is desirable in patients in whom a thoracoplasty is not indicated.

4. A little more precaution than that necessary for a phrenic neurectomy or scaleniotomy is required if the anatomic structures of this region are kept in mind.

After our paper was submitted for publication, Dr. R. W. McNealy[9] used a technic, based on similar general principles, for the resection of the stellate ganglion in patients who need sympathectomy.

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CHILDBIRTH FOLLOWING THORACOPLASTY*†

Report of a Case

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Wallum Lake, R. I.

CHILDBIRTH in tuberculous patients who have undergone thoracoplasty is of comparatively rare occurrence if the paucity of reported cases is to be relied upon. The compatibility of childbearing with subsequent good health, in women whose tuberculosis has been treated by thoracoplasty, seems not sufficiently recognized. After a careful search of the literature, I have been able to find only seven such cases on record, the first being reported by Saye[1] in 1926. In none of these cases were there any reported complications during parturition. All went to full term except one case, in which the pregnancy was terminated at seven months.[5] There was no progression of the pulmonary lesion postpartum. All the children were living and well at the time these cases were reported.

In one case,[1] thoracoplasty was done during the course of pregnancy. In the other cases delivery took place at varying intervals after thoracoplasty; the earliest, after an interval of twenty months.[3] In two cases the interval was three years[3, 5]; in the other three instances, the interval was four and a third,[4] four and a half,[2] and five years.[5]

Following is another case which I wish to add to the list:

M. C., white woman, aged twenty-one years. First admission July, 1925. Discharged November, 1925. Symptoms of cough, expectoration, loss of weight and fever came on shortly after birth of her first child in March, 1925. One month before admission to the State Sanatorium, Wallum Lake, she developed right-sided pleurisy, night sweats, and fever which went as high as 105° F. Physical examination and roentgenogram showed an extensive exudative lesion with multiple excavation involving the right upper lobe. Weight dropped from her usual 110 pounds to 90 pounds. Sputum, constantly positive, varied daily between 60 and 150 grams. After staying at the Sanatorium for four months, she left against advice, unimproved.

Second admission February, 1926. Discharged October, 1931. On this admission, symptoms, physical examination and roentgenogram (Fig. 1) showed the patient to be in poor condition. She had symptoms of laryngeal and intestinal tuberculosis. Right-sided pneumothorax was attempted in February, 1926, but failed because of pleural symphysis. A number of copious pulmonary hemorrhages occurred in February, 1928. The beginning of her clinical improvement (about February, 1928) was coincident with onset of another pregnancy. Her weight had risen to 117 pounds. The daily amount of sputum had decreased to 30 grams, but still remained positive for tubercle bacilli. In August, 1928, she went through an uncomplicated parturition.

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†From the State Sanatorium.

On her return from the Providence Lying-In Hospital in September, 1928, symptoms of active tuberculosis were more marked. The tuberculous activity subsided, however, by December, 1929, when she delivered a third child after an uncomplicated labor. After a temporary exacerbation of symptoms, she again improved. In August, 1930, a right phrenicectomy was done; for two and a half months subsequently, dyspnea and gradual progression of the disease were noted. Symptoms were entirely alleviated by right-sided thoracoplasty done in November, 1930. The operation was performed in two stages by Dr. Wyman Whittemore at The Massachusetts General Hospital. Recovery was uneventful. Upon discharge from the Sanatorium she had no cough, no expectoration, she felt well and had gained in weight up to 128 pounds. Prior to thoracoplasty, monthly sputum examinations were almost always positive; following thoracoplasty, sputum became negative. Since the thoracoplasty,

Fig. 1.—Roentgenogram taken February, 1926. Exudative tuberculosis with multiple excavation throughout the right lung. Left lung shows infiltration in the central third.

patient has had no clinical evidence of intestinal tuberculosis, nor of laryngeal tuberculosis except for persistent hoarseness.

Further Course.—Patient continued to gain strength and soon assumed full household duties. She has had no recurrence of symptoms. She gave birth, at full term, to a fourth child, two and a half years after thoracoplasty (May, 1933). Nitrous oxide and oxygen anesthesia was administered to relieve labor pains. Delivery, which was noninstrumental, lasted two hours.

At the time of writing, the baby is seven months old, is in good health, and weighs 17 pounds. Feeding has been artificial. The mother is slowly gaining back her temporarily lost weight (now 100 pounds) and strength. Repeated sputum examinations in the early part of January, 1934, showed one positive for tubercle bacilli: she had had an upper respiratory infection lasting one week. She is now

doing her own housework and caring for the baby. The latest roentgenogram (Fig. 2) taken November, 1933, shows good surgical collapse of the right lung and a slight inactive lesion in the contralateral lung.

SUMMARY

A case is recorded of childbirth taking place two and a half years following thoracoplasty. The patient had gone through three uncomplicated pregnancies during the course of her tuberculosis prior to thoracoplasty. After each parturition there was an exacerbation of symptoms which gradually subsided. Since thoracoplasty and the pregnancy following it, the patient has been in relatively good health.

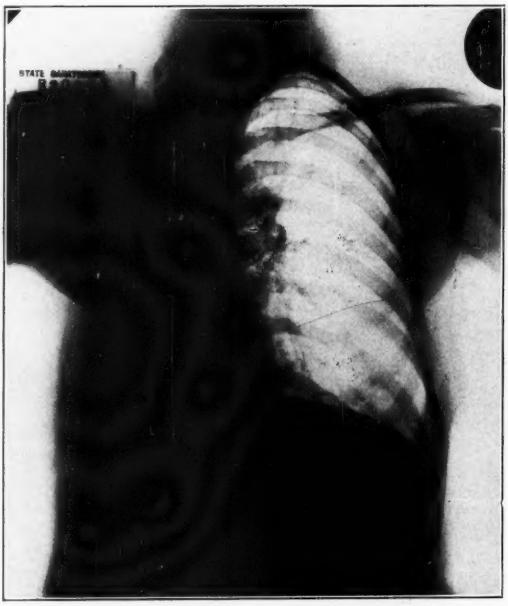

Fig. 2.—Roentgenogram taken November, 1933. Right: Thoracoplasty. Left lung shows fibrotic changes in apex and central third.

The author wishes to express his gratitude to Dr. A. A. Karan for his help in preparing the manuscript.

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DIAPHRAGMATIC HERNIA IN A RABBIT*

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THE incidental finding of a diaphragmatic hernia in an experimental rabbit seemed worthy of record in respect to comparative biology. A cursory review of the literature reveals no reference to the occurrence of diaphragmatic hernia in animals.

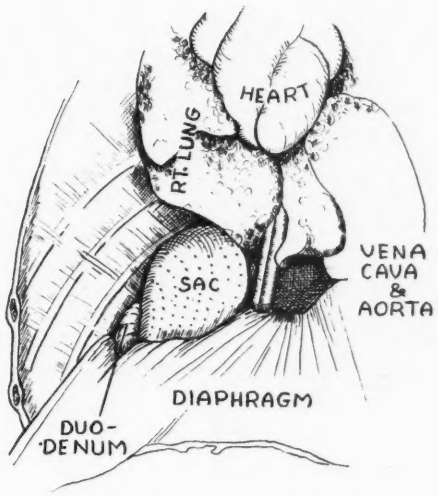

Fig. 1.—The duodenum has escaped through the incision made into the sac.

Upon opening the right pleural cavity of a rabbit for the purpose of producing a traumatic pleuritis my attention was directed to a smooth lump the size of an English walnut projecting from the dome of the diaphragm. Ten c.c. of boiling water were instilled into the pleural cavity; the chest was closed, and the mass was not investigated until the animal died three weeks later.

The mass proved to be a hernial sac which seemed to be continuous with the diaphragm and consisted of fibrous tissue with serous cover-

*From the Departments of Surgery and Clinical Research, University of Nebraska School of Medicine.
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ing and lining. It contained a lobe of the liver and a large loop of the duodenum. The hernial ring measured 2.5 cm. in diameter and was

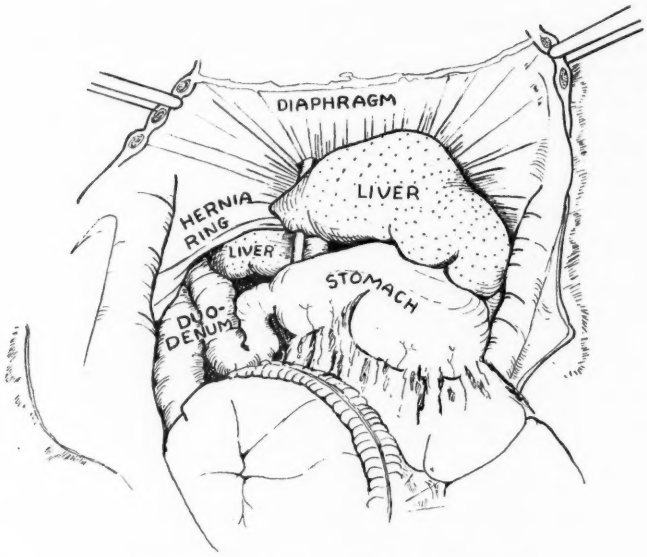

Fig. 2.—Hernia viewed from below diaphragm.

situated slightly posterior and to the right of the caval perforation. The autopsy findings are illustrated diagrammatically in Figs. 1 and 2.

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

A. Mouchet. Traumatic Chylothorax. J. de chir. September, 1933.

The diagnosis is made by analysis of the fluid which consists of chyle and is sometimes strained by blood; it may be in one or both pleural cavities. Diagnosis is important because of the pathologic and therapeutic problems which arise.

The important anatomical features involved are the relation of the mediastinal pleura along the lower end of the azygo-esophagic sac. This concerns the right thorax mainly, but in the superior thoracic portion of the duct, "sus aortic" it is closer to the left mediastinal pleura. Rupture of the duct without lesions of the bones is rare. The more common bone lesions are: fracture of the inferior dorsal spine when the duct is torn in its lower portion and fracture of the clavicle when it is injured higher up.

Clinically, there are two types of chylothorax, depending upon whether the pleural cavity is open or closed. The symptoms in a closed chylothorax, which is the commonest kind, are the presence of fluid from two to five days after traumatism, shock at the beginning of the collection, rapid establishment and rapid reestablishment of the fluid, marked dyspnea, and symptoms of starvation, such as malnutrition and asthenia. Oliguria is frequent, and there is a pouring out of fat into the pleural cavity after ingestion of fats into the stomach.

The open thorax usually follows stab wounds or gunshot wounds. Such wounds are usually situated at the base of the neck. Closed chylothorax usually follows automobile accidents or similar violent traumatism to the chest, although one case is on record in which the patient had suffered a mild traumatism after colliding with a door. Occasionally it follows operations in the lower portion of the neck. The treatment indicated is removal of the fluid, and this may or may not be followed by injection of air into the chest. Intravenous injection of the fluid withdrawn has been done but has not proved effective. Thoracotomy is beneficial in that the duct may be reestablished by adhesions following the surgical intervention. Ligature of

the canal or packing in the cervical region of the canal is indicated in some cases. If infection occurs, thoracostomy with drainage is indicated.

Dr. L. Rey, Havana.

C. Xalabarder. Thoracoplasty in the Medical Sanatorium. Rev. méd. de Barcelona **19**: 433, 1933.

The author reports a study of thirty thoracoplasty operations in the last two years without mortality in the eight weeks following operation. He places importance on the selection of cases and says that patients must have previous treatment in a sanatorium months before operation. The improvement of the general condition, fever and expectoration is important.

Many cases were treated by pneumothorax and sodium thiosulphate, which provoked resorption in a great many of the lesions of the opposite lung. He cites the case of a patient with a positive Wassermann who had been treated with arsenicals previous to the operation. He believes that preoperative treatment is important: 60 grams honey, insulin, 40 drops of hepatine, 0.2 gram of camphor daily, digitalis, and rest in bed. He also makes the clinical tests to ascertain the condition of the heart and kidneys. He gives special attention to the oculocardiac reflex in relation to the use of eserine with general anesthesia. He describes the technic he uses, the principal features being: ether chloroform anesthesia, time of operation from thirty-five to seventy minutes; he recommends the low Frauchaud incision because it gives easy access to the first rib and also gives good results. The postoperative treatment is as follows: hot bed for the patient, and 1 c.c. coramine just after operation. The first night 1 c.c. pantopon is administered. He reports the absence of postoperative accident and a temperature elevation that falls to normal after from eight to ten days. He reports late complications in only two cases, one of which presented a pneumonic focus in the base of the lung on the side of operation. The other patient developed a cavity on the opposite side, where a cavity had been long ago; after being treated with sodium thiosulphate it healed.

He mentions operative mortality and holds it to that which occurs the first days after operation and in relation with the operation. The time differs with authors: Sauerbruch, four weeks; Berard, three weeks; Bull, eight weeks. In regard to the mortality in relation to the different technics: Brauer-Friedrich 30 per cent to 40 per cent; Sauerbruch (paravertebral) 17 per cent. Causes of mortality depend on: (1) the patient; (2) the technic; and (3) the site of operation.

The patient: Mortality is higher in men (13 per cent) than in women (9.7 per cent). Site: Thoracoplasty on the right side is more danger-

ous (18.6 per cent) than on the left side (5.1 per cent). The general condition of the patient is important. Previous treatment by sodium thiosulphate (pure) sanocrysin is advised.

Technic: In one stage, 23.8 per cent. In various stages, 7.9 per cent. The author prefers general anesthesia, but believes that it is contraindicated in cases where the expectoration is 80 c.c. (according to Berard), 50 c.c. (according to Bull), 20 c.c. (according to Brauer). He operates on patients with more than these amounts of expectoration, under general anesthesia, but in the Trendelenburg position.

Preoperative and postoperative treatment is important as already stated, and the best results are obtained in those patients who have had treatment in a sanatorium.

Dr. L. Rey, Havana.

Book Review

SURGERY OF THE THORAX, by T. Holmes Sellors, M.Ch., M.A., B.M. (Oxon.), F.R.C.S. (Eng.); 140 illustrations including 11 plates in color, Constable and Company, Ltd., London, 1933.

This book of 476 pages of text will be found to be a useful summary of surgical procedures as applied to thoracic conditions. The book is much more a description of operative methods than it is a discussion of diagnosis and pathology. Apparently the author's intent has been to prepare a book which would be useful chiefly for the general surgeon who wishes to perform thoracic operations; although in the introduction he speaks about the advisability of a cooperation between the physician and the surgeon in this field. One can obtain a very good idea of the surgical procedures by reading this book, but one cannot find much discussion of the etiologic, pathologic and diagnostic features of the various conditions. The author states frankly in his introduction that because the British literature has been influenced to a very great extent by American writings it has been his purpose to bring to the attention of the English speaking world the work of the German and Scandinavian thoracic surgeons. The book is obviously based on a very limited personal experience in this field. Many of the illustrations are taken from Sauerbruch's monumental *Chirurgie der Brustorgane*. The author, however, does not place the American work at any disadvantage. The bibliography of twenty-seven pages contains most of the best American work.

In general the subject matter and the manner of presentation can be praised highly. Certain statements, however, strike the reviewer with some surprise. For example, one is amazed to find on page 92 the following paragraph: "American literature is singularly devoid of the use of lipiodol as compared with English and Continental experience, and the chief references to examinations of this nature consist in descriptions of blowing an opaque powder (bismuth) into the bronchi through an endoscope. This procedure appears needlessly drastic when compared with lipiodol administration." Possibly Mr. Sellors has obtained the impression of the lack of use of lipiodol because of the unwillingness on the part of the journals published by the American Medical Association to permit the use of the word. Since various subterfuges such as "iodized oil" have necessarily come into use in the American literature, it is not surprising that a foreigner who has never visited our thoracic clinics would get the impression from a perusal

of the literature that lipiodol is not much used in this country. The author is evidently not familiar with some of the excellent results which have been obtained by apical thoracoplasty alone in tuberculosis, because on page 345 he states that "the only efficient and effective method is to produce collapse of the whole lung." On page 363 he also states "the base should, therefore, always be collapsed first before the contents of upper zone cavities are forced out by rib resection." It seems rather curious also that in a discussion of the treatment of thymus enlargements no mention is made of the use of radiation. Operative methods which are now obsolete in this country are apparently recommended by the author on pages 388 and 389. The interesting suggestion is made on page 130 that the word "synechiotomy" should be used in preference to the more usual term of "intrapleural pneumolysis." While on the subject of terminology the reviewer would much prefer to use the word "sclerotic" instead of the word "cirrhotic" which the author uses on page 340. The word cirrhotic which was applied to a certain disease of the liver was used originally in its true sense because it meant a yellow color which the liver had. It had no reference to a thickening of the liver. There is no etymologic justification, therefore, for speaking of cirrhotic lungs unless for some peculiar reason they might have a yellow color.

The book is written in an interesting manner, and it will be found to be an excellent addition to the library of any one who is interested in thoracic surgery.

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Seventeenth Annual Meeting of the American Association for Thoracic Surgery

Boston, May 31, June 1 and 2, 1934

The seventeenth annual meeting of The American Association for Thoracic Surgery will be held in Boston on Thursday, Friday and Saturday, May 31, June 1 and 2, 1934. Those interested in the subject, or related subjects, are welcome to attend the meetings. It is not necessary to be a member of the Society in order to attend. Please make your reservations through Dr. Edward Churchill, Massachusetts General Hospital, Boston.

The program this year, as usual, will be rather inclusive and will touch upon many of the different phases pertaining to the surgery and the physiologic problems concerning the thorax. The papers will deal with clinical studies and with original research problems.

The outline of the program is for a full day of formal discussion on Thursday, May 31. The formal program will be extended to Friday, June 1, and will occupy one-half of the day. The remaining half day will be devoted to the research work which is being carried on in Boston in the various clinics and hospitals. Saturday, June 2, will be given over to operative clinics and clinical presentations. This arrangement will allow for three full sessions for the reading of papers and three full sessions for clinical presentations and local research.